This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
2 45 0309 0405

LANE

MEDICAL LIBRARY

SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885

BOUND WITH FUNDS GIVEN IN HONOR OF

A. L. BLOOMFIELD, M.D.

LANE LIBRARY. STANFORD UNIVERSITY

SUPPLEMENT

TO

ZIEMSSEN'S CYCLOPÆDIA

OF THE

PRACTICE OF MEDICINE

EDITED BY
GEORGE L. PEABODY, M.D.
Instructor in Pathology and Practice of Medicine, College of Physicians and Surgeons, New York;
Pathologist and Medical Registrar to the New York Hospital.

N. Y. Academy of Medicine
LIBRARY,
12 West 31st St., New York

NEW YORK
WILLIAM WOOD AND COMPANY
27 GREAT JONES STREET
1881

K

Copyright, by
WILLIAM WOOD & COMPANY,
1881.

STEAM PRESS OF
H. O. A. Industrial School,
187 & 189 E. 70th St.

pL.
11

CONTRIBUTORS TO THIS VOLUME.

EORGE B. SHATTUCK, M.D., of Boston.
Physician to Carney Hospital; Physician to Out-Patients, Boston City Hospital; Physician to Boston Dispensary.

EORGE M. STERNBERG, M.D.
Surgeon U. S. Army.

ANCIS P. KINNICUTT, M.D., of New York.
Visiting Physician to St. Luke's Hospital; Physician to Out-Patient Department, New York Hospital.

FORCHHEIMER, M.D., of Cincinnati.
Prof. of Physiology, and Clinical Prof. of Diseases of Children, Medical College of Ohio.

ANK P. FOSTER, M.D., of New York.
Physician for Diseases of Women to the Out-Patient Department, New York Hospital.

ENRY M. LYMAN, M.D., of Chicago.
Prof. of Physiology and of Diseases of the Nervous System, Rush Medical College, Chicago.

LEWIS SMITH, M.D., of New York.
Clinical Prof. of Diseases of Children in Bellevue Hospital Medical College.

MES NEVINS HYDE, M.D., Chicago.
Prof. of Skin and Venereal Diseases, Rush Medical College, Chicago.

MES LAW, F.R.C.V.S., of Ithaca, New York.
Prof. of Veterinary Science in Cornell University, Ithaca.

E. SATTERTHWAITE, M.D., of New York.
President of the New York Pathological Society; Pathologist to St. Luke's and the Presbyterian Hospitals.

ORGE M. LEFFERTS, M.D., of New York.
Clinical Prof. of Laryngoscopy and Diseases of the Throat, College of Physicians and Surgeons, New York; Consulting Laryngoscopic Surgeon to St. Luke's Hospital.

SOLIS-COHEN, M.D., of Philadelphia.
President of the American Laryngological Association; Lecturer on Larnygoscopy and Diseases of the Throat and Chest in Jefferson Medical College; Physician to the Jefferson College Hospital, and to the German Hospital.

EORGE M. GARLAND, M.D., of Boston.
Prof. of Thoracic Diseases, University of Vermont.

27084

FRED'K C. SHATTUCK, M.D., of Boston.

Visiting Physician to the House of the Good Samaritan; Physician to Out-Patients, Massachusetts General Hospital; Clinical Instructor in Auscultation and Percussion in Harvard University.

T. M. ROTCH, M.D., of Boston.

Physician to the Boston Dispensary; Clinical Instructor in Diseases of Children in Harvard University.

A. T. CABOT, M.D., of Boston.

Attending Surgeon to Carney Hospital.

ELBRIDGE G. CUTLER, M.D., of Boston.

Visiting Physician to Carney Hospital; Physician to Out-Patients, Massachusetts General Hospital.

ROBERT F. WEIR, M.D., of New York.

Lecturer on Genito-Urinary Diseases at the College of Physicians and Surgeons; Attending Surgeon to the New York and Roosevelt Hospitals, etc.

REGINALD H. FITZ, M.D., of Boston.

Prof. of Pathological Anatomy, Harvard University.

S. G. WEBBER, M.D., of Boston.

Clinical Instructor in Diseases of the Nervous System in Harvard University; Visiting Physician to Department for Diseases of Nervous System, City Hospital, Boston; Consulting Physician to Carney Hospital.

L. PUTZEL, M.D., of New York.

Physician to the Clinic for Nervous Diseases, Bellevue Hospital Out-Door Department; Visiting Physician for Nervous Diseases, Randall's Island Hospital; Pathologist to the Lunatic Asylum, B. I.; Curator to Charity Hospital, etc.

R. VAN SANTVOORD, M.D., of New York.

Visiting Physician to the Work-House and Alms-House Hospitals.

JAMES J. PUTNAM, M.D., of Boston.

Physician to Out-Patients, Massachusetts General Hospital; Lecturer on Diseases of the Nervous System, Harvard University.

A. B. BALL, M.D., of New York.

Attending Physician to St. Luke's Hospital.

CHARLES E. HACKLEY, M.D., of New York.

Visiting Physician to the New York Hospital and Trinity Infirmary.

HENRY N. HEINEMAN, M.D., of New York.

Visiting Physician to the Mt. Sinai Hospital, and the Out-Patient Department, New York Hospital; Assistant Pathologist to Roosevelt Hospital.

H. M. BANNISTER, M.D., of Chicago.

EDWARD S. WOOD, M.D., of Boston.

Prof. of Chemistry, Medical College, Harvard University.

PREFACE.

So valuable a work as *v. Ziemssen's Cyclopædia* ought not to be allowed to grow old, and the object of this volume is to remove from it the few traces of time that the last few years have produced.

The scope of this book is indicated by its name. It may be well to call attention to the fact that it is intended to cover only those subjects treated in the American edition of *v. Ziemssen's Cyclopædia,* of which work some of the volumes are now five or six years old, others being quite recent. It has been the aim of the writers to give here a concise account of the progress made in the various departments of medicine during the time that has elapsed since the several volumes of the Cyclopædia were published, each of the subjects treated being brought up to the date of the present volume. The arrangement as to bibliography, captions, paragraphs, etc., is essentially the same as in the original work. It will be observed that some of the subjects treated in the Cyclopædia have not been mentioned in this volume. They have been omitted because nothing of importance has since appeared in regard to them.

It is believed that, independently of its connection with the Cyclopædia, as a volume giving the results of recent advances in medical science, the present book will be acceptable to the profession.

The materials have been derived from original observation and from the medical literature of all countries. The labor necessary in making such a digest is enormous; and it is believed that the names of the writers who have undertaken the work in their various departments will be ample guarantee for the fidelity with which it has been done.

GEORGE L. PEABODY.

TABLE OF CONTENTS.

PAGE

l Fever 3
ıg Fever 25
gue 33
Fever 45
nd Diphtheria 77
a 99
.... 103
.... 105
ox 115
l Diseases 131
.... 139
las 145
-Spinal Meningitis 151
.... 167
s 187
.... 193
ıobia 207
ısis 214
al Parasites 217
of the Larynx 227
of the Nose 259
of the Pharynx 264
of the Pleura 271
of the Lungs 281
of the Heart 349
of the Pericardium 361
of the Arteries 371
of the Veins 380
of the Lymphatics (including Chyluria) 380
of the Stomach 389
of the Intestines 400
of the Spleen 403
of the Bladder 431
of the Urethra 447
ıce 456
of the Liver (including Jaundice, Cholecystotomy, and Hypertrophic hosis) 467
of the Female Sexual Organs 477

PAGE
Diseases of the Peripheral Cerebro-Spinal Nerves 511
Diseases of the Brain 537
Diseases of the Medulla Oblongata 560
Diseases of the Spinal Cord 567
Vaso-Motor and Trophic Neuroses 603
Diseases of the Kidneys 655
Rheumatism 711
Arthritis Deformans 727
Gout 730
Osteomalacia 732
Slight Disorders caused by Catching Cold 736
Anæmia 737
Essential Pernicious Anæmia 750
Corpulence 752
Scrofulosis 753
Malignant Lymphoma 761
Idiopathic Adenitis 763
Diabetes Mellitus 767
Diabetes Insipidus 779
Hæmophilia 785
Scurvy 789
Morbus Maculosus Werlhofii 798
Toxicology 801

TYPHOID FEVER.

RELAPSING FEVER.—PLAGUE.

BY

GEORGE B. SHATTUCK, M.D.,

OF BOSTON,

PHYSICIAN TO THE CARNEY HOSPITAL; PHYSICIAN TO OUT-PATIENTS, BOSTON CITY HOSPITAL; PHYSICIAN TO BOSTON DISPENSARY.

TYPHOID FEVER.

BIBLIOGRAPHY.

AGENBACH, E.: Epidemiologisches aus Basel. Jahrbuch für Kinderheilkunde,
, IX., S. 50, 1875.—VIRCHOW: Typhus und Städte-Reinigung. Deutsch. med.
h., 1876. –RUTENBERG: Abkühlung des Körpers vom Darme aus. Deutsch.
Wochenschr., II., 19, 1876.—v. ROTHMUND: Aetiologie des Typhus Abdomi-
. Bayer. ärztl. Intell. Blt., XXII., 7, 1875.—VALLIN: La Fièvre Typhoïde et
ppe d'eau souterraine de Paris. Gaz. hebd., 2 Sér. XIII., p. 785, 1876.—DOE,
.: Efficacy of Cold Water Treatment in Typhoid Fever. Boston Med. and
. Journ., XCIV., p. 244, 1876.—Propagation of Enteric Fever by Infected
: the Eagley Outbreak. Lond. Practit., XVII., p. 225, 1876.—LOW, R. BRUCE:
nteric Fever ever Spontaneously Generated? Brit. Med. Journ., I., p. 659,
– FEREOL, S.: Sur le traitement de la fièvre typhoïde par les bains froids.
n Médicale, XXII., December, 1876.—Discussion à la Société Médicale des
taux. Union Médicale, XXII., 1876.—BAHRDT, R.: Experimentelle Unter-
ungen über die Uebertragbarkeit des Typhus Abdominalis auf Thiere.
iv der Heilkunde, XVII., p. 156, 1876.—WOODWARD, J. J.: Typho-malarial
r: Is it a Special Type of Fever? Trans. Internat. Med. Con., p. 305, Phila-
hia, 1876.—GOLTDAMMER: Kaltwasserbehandlung des Ileotyphus, Bericht
die Resultate der Kaltwasserbehandlung des Ileotyphus im Krankenhaus
anien in Berlin. Deutsch. Archiv f. klin. Med., XX., p. 52, 1877.—PROF.
R: Les bains froids coup sur coup dans la fièvre typhoïde. Union Médicale,
II., 3 s., 1877.—RAYNAUD, M.: Du traitement de la fièvre typhoïde par les
s froids. Bull. génér. de therap., p. 433, 1877.—ALBU: Typhus und Grund-
ergang in Berlin. Berlin. klin. Wochenschr., XIV., S. 422, 1877.—LIBER-
s: De l'influence des bains froids sur les complications laryngiennes de la
e typhoïde. L'Union Médicale, XXIII., 3 s., p. 298, 1877.—EDES, R. T.: The
-Water Treatment of Typhoid Fever. Boston City Hosp. Rep., p. 205, 1877.
RPHY, SHIRLEY F.: Antipyretic Treatment of Enteric Fever. London Lancet,
. 791, 1877.—DUDGEON, JOHN: On the Sanitary Condition of China. Sewage,
nage, and Typhoid Fever. Glasgow Med. Journ., IX., p. 179, 1877.—CABELL,
.: Etiology of Enteric Fever. Trans. Am. Med. Assoc., p. 411, 1877.—
ATOR: Ueber die therapeutischen Wirkungen des Salicins. Berlin. klin.
henschr., XIV., S. 182, 1877.—STONE: Etiology of Typhoid Fever. Tr. Maine
. Assoc., p. 383, 1878.—LETZERICH: Experimentelle Untersuchungen über
hus Abdominalis. Arch. f. exper. Path. und Pharm., IX., S. 312, 1878.—HOPE,
.: Typho-Malarial Fever. Trans. Tennessee Med. Soc., p. 63, 1878.—Treatment
yphoid Fever in Bellevue Hospital. New York Med. Record, XIV., p. 366,
.—CLARK, ALONZO: Lectures on Fevers. N. Y. Med. Record, XIII., pp. 261,
301, 1878.—PEPPER, W.: Lecture on Typhoid Fever. Phil. Med. Times, VIII.,
3, 1878.—THOMSON, WILLIAM: Typhoid Fever, its Cause and Extent in Mel-
ne. Melbourne, 1878.—BALLARD: Enteric Fever at Ascot. Sanitary Record,
p. 277, 1878, X., p. 35, 1879.—COLLIE A.: Value of Cold Baths in Enteric Fever.
d. Practit., XXI., p. 347, 1878.—MARKS: The Prevention of Typhoid Fever.
rt of Board of Health of Wisconsin, III., p. 22, 1878.—GAIRDNER, W. T.: Cau-
s in respect of the so-called "Antipyretic Treatment" in Specific Fevers. Glas-
Med. Journ., X., pp. 416, 499, 1878.—LIEBERMEISTER: On the Antipyretic

Treatment of Specific Fevers. Glasgow Med. Journ., X., p. 449, 1878.—SMITH, F. G.: Treatment of Hyperpyrexia. Lond. Prac., XX., p. 20, 1878.—GEISSLER: Bericht über den Typhus. Schmidt's Jahrbücher, CLXXVII., S. 57, 1878.—IMMERMANN: Ueber Prophylaxe von Typhus-Recidivien. Corr. Bl. f. Schweiz. Aerzte, Nos. 23 and 24, S. 697, 737, 1878.—HOMOLLE: La Fièvre Typhoïde, Revue Générale. Rév. des Sciences Méd., XI., p. 769, 1878.—WALDER: Ueber die Typhusepidemie von Kloten. Berlin. klin. Wchnschr., 1878.—KLEIN, E.: Experimental Contribution to the Etiology of Infectious Diseases. Quar. Journ. Microscop. Sciences, XVIII., N. S., p. 170, 1878.—EWART, J. COSSAR: Life History of Bacillus Anthracis. Quar. Journ. Mic. Sci., XVIII., N. S., p. 161, 1878.—KING: Etiology of Typhoid Fever. Med. Times and Gaz., I., p. 118, 1879.—JACOBI, A.: Typhoid Fever in Infancy and Childhood. New York Med. Record, XVI., pp. 385, 411, 1879.—SOYKA: Boden. Real-Enclyclopädie der gesammten Heilkunde. Wien, 1879.—JENNER, SIR WM.: Treatment of Typhoid Fever. London Lancet, II., p. 715, 1879.—SMYTHE, G. C.: Antipyretic Treatment of Typhoid Fever. American Practit., XIX., p. 7, 1879.—BILLINGS, J. S.: Buck's Hygiene and Public Health, Introduction, I., New York, 1879.—THOMSON, W.: Typhoid Fever; Contagious, Infectious, and Communicable. Brit. Med. Journ., I., p. 343, 1879.—MURPHY, SHIRLEY F.: Etiology of Enteric Fever. Brit. Med. Journ., I., p. 581, 1879.—COLLIE, A.: Etiology of Enteric Fever, with an Account of Nineteen Cases which occurred in the Homerton Fever Hospital. Brit. Med. Journ., I., pp. 341, 385, 424, 1879.—STEDMAN, C. E.: 150 Cases of Typhoid Fever in Boston City Hospital. Boston Med. and Surg. Journ., CI., p. 499, 1879.—SKREZCKA, C.: Typhus Morbilität in Berlin. Vrtljschr. f. Gerichtl. Med., Berl., N. F., XXX., S. 143, 1879.—The Recent Epidemic of Enteric Fever in the Caterham Valley. Brit. Med. Journ., I., p. 669, 1879. Ibid. Med. Times and Gaz., I., p. 505, 1879.—WALDER: Typhoid Epidemic Originating in Diseased Meat. Med. Times and Gaz., I., p. 149, 1879.—MASON, A. L.: Reports on Progress in the Theory and Practice of Medicine. Bost. Med. and Surg. Journ., C., pp. 426, 459, 1879.—CABELL, J. L.: Salicylic Acid in the Treatment of Enteric Fever. Virginia Med. Monthly, V., p. 424, 1879.—FAUNTLEROY: Typhoid Fever; its Etiology and Treatment. Virgin. Med. Mon., V., p. 415, 1879.—FRANKLAND, PROF.: Sewage. Journ. Soc. of Arts, XXVII., 1879.—COLLIE, A.: Etiology of Enteric Fever. Brit. Med. Journ., I., p. 84, 1880.—ALISON: Etiologie de la fièvre typhoïde dans les Campagnes. Archiv. Gén. Méd., Jan., Fev., Mars, Paris, 1880.—FOLSOM, C. F.: Suggestions as to the Causes of Typhoid Fever. Bost. Med. and Surg. Journ., CII., p. 227, 1880.—EWART, JOSEPH: Enteric Fever in India; with some Observations on its Probable Etiology in that Country. Med. Times and Gaz., I., p. 231, 1880.—CASWELL, E. T.: The Alcohol Question from a Medical Point of View. Trans. Rhode Island Med. Soc., Providence, 1880.—HOFF, J. V. R.: Typho-Malarial Fever; the so-called "Mountain" Fever of the Rocky Mountain Region. Am. Jour. Med. Sciences, Jan., 1880.—ZUELZER: Abdominal Typhus. Real-Encyclopädie der gesammten Heilkunde, S. 11, Wien, 1880.—GREENFIELD, W. S.: Lectures on some Recent Investigations into the Pathology of Infectious and Contagious Diseases. Lond. Lancet, I., pp. 83, etc., 1880.—CAMPBELL, A.: Spread of Typhoid Fever to Animals. Brit. Med. Journ., I., p. 555, 1880.—CARPENTER, A.: A Consideration of some of the Fallacies which are based upon a Narrow View of the Germ Theory. London Lancet, I., p. 79, 1880.—BALLARD, E.: Observations on some of the Ways in which Drinking Water may become Polluted with the Contagion of Enteric Fever. London Lancet, I., p. 82, 1880.—CAYLEY: Pathology and Treatment of Typhoid Fever. Brit. Med. Journ., I., pp. 391, etc., 1880.—CARMICHAEL, NEIL: An Experimental Investigation into the Trap and Water-Closet System, and the Relation of the same to Sewage Products, Gaseous and other. Glasgow Sanitary Journal, N. S., IV., No. 49, 1880.

The last four or five years have been by no means inferior to their edecessors in the amount of attention and interest which this subject s demanded at the hands of professional and scientific men. In fact, vestigations, observations, and theories have increased rather than minished. As might have been anticipated, however, there is little or thing to add to the pathology of the disease, and the work which we ve to record, whether in the form of experiment, observation, or theory, ls almost entirely under the divisions of etiology and treatment.

Of work, there has been much, of advanced posts in the unexplored debatable regions which have been taken possession of in the name of curate knowledge and securely held against all assault, until a fair title s been established for science, we can point to very few.

There is still much discussion as to whether typhoid fever is a contaous disease, and as to whether it has at times a *de novo* origin. The estion of contagion continues to be obscured by the varied uses of the rd contagious. If all writers would agree as to the subject, there would less conflicting differences of opinion. The poison of typhoid fever, is generally agreed, is probably particulate, neither liquid nor gaseous. e classification of the disease as miasmatic-contagious is perhaps ll the best possible, in so far as it expresses the more usual manner of propagation. In formulating the statement that the disease is not ntagious, it should be remembered that strict personal contact is meant, d if what we are able to affirm is to be limited to so small and unimrtant a point, it might be as well to express the relation of typhoid er to some other zymotic diseases from the stand-point of a contagium a matter of degree rather than of kind. If we say that typhoid fever never directly transmitted from person to person, the statement must qualified by the admission that at times, even if rarely, the stage of velopment of the poison outside the human body, if such be necessary a further reproduction of the disease, occupies a very short period, rhaps some hours only. Otherwise we must await further knowledge explain a by no means small class of carefully observed cases. In the nited States at least, there no longer exists any confusion between typhus d typhoid fevers to complicate a problem which is sufficiently difficult thout it. A few points, among many, which affect the question of ntagion may with propriety be touched upon.

Jacobi, contrary to the usually received opinion, maintains that phoid fever is not rare either in infancy or childhood. Dr. *Wiltshire*, St. Mary's Hospital, London, after reporting a case of enteric fever in infant six months old, expresses a suspicion that this disease is not frequently overlooked in young children; "gastric fever," "infantile nittent fever," "low fever," "slow fever," being assigned as the cause illness. Others also suggest that one reason why adults are at times posed with impunity is, that having had the disease in infancy they e protected. These suggestions again remind us of that border-land of phoid, where we find "cess-pool" fever (*Clark*), "typho-malarial" ver (*Woodward*), "slow" fever, all of which by complicating observans confuse our views, not only as to etiology, but also as to contagion.

Constant observations, moreover, only serve to confirm the tenacity of life of the typhoid poison under favorable circumstances for long periods, its activity even when extremely diluted (Caterham epidemic), and its transportability in bedding, clothes, etc. These facts must be kept in mind in forming opinions as to contagion.

Dr. *Collie*, of the Homerton Fever Hospital, a strong contagionist in the strict sense of the word, has published a number of cases occurring at Homerton among the nurses and attendants which he is convinced arose from from *fresh* enteric stools. He thinks their age—so large a proportion being over thirty—has some bearing upon the small percentage of physicians and attendants in hospitals who contract this disease. The greatly diminished liability of persons over thirty years of age scarcely needs further illustration. Out of 4,928 total admissions of enteric fever to London, Stockwell, and Homerton Fever Hospitals, 3,915 were under 25 years of age, 4,352 under 30; out of 1,436 admissions to Homerton between 1871–1878, inclusive, 1,158 were under 25, 1,279 under 30 years. *v. Rothmund* reports nine cases of typhoid fever among the attendants in an army hospital within nine months, although the sanitary arrangements were especially good in his estimation.

Mr. *Murphy*, in discussing Dr. *Collie's* conclusion that the twenty cases of enteric fever, reported by him as occurring among the attendants at the Homerton Fever Hospital, had their origin in the fresh stools of typhoid patients, shows that between the years 1855–1878 inclusive, five thousand five hundred and sixty-nine enteric patients were located in the London Fever Hospital, and during that time nineteen persons engaged in the Hospital were attacked, ten of these were in no way connected with enteric fever patients or wards, and one was a laundry-maid. Estimating that about the same number of nurses are engaged in the two hospitals, he calculates that if the Homerton Hospital were to maintain the same number of attacks among attendants it has done for the past six and a half years, it would in twenty-four years have nearly sixty of its nurses attacked. He concludes that such a difference must be due, not to causes common to both hospitals, but to some difference in the structure of the hospitals themselves; and the only difference suggesting itself is the drainage. Dr. *Collie*, in his reply, reasserts his former view, and exculpates the drainage which had been thoroughly investigated. He explains the different results given by the statistics of the two institutions, by a dffierence in the age of the nurses; those attacked at Homerton being all under thirty, except one of that age. Unfortunately we do not know the proportion of nurses at the London Hospital under thirty years of age.

Sir *Thomas Watson* says: I hold that, like small-pox, etc., the disorder may be taken by breathing emanations from the sick person's body or bed, and that scrupulous care should be taken in all cases not to inhale the poison in that way.

The practical point which the present state of knowledge in regard to the contagion of typhoid fever should cause to be emphasized is the immediate disinfection of the alvine discharges of typhoid patients, or even of those suspected of this disease. Without denying the elimination and absorption of the poison by other surfaces than that of the bowel, the evidence in regard to the propagation of the disease does not, to our mind, demand that isolation be practised with it as with small-pox, at least where there is no overcrowding and good ventilation can be secured. Greater precautions doubtless are necessary when dealing with this malady in small, ill-ventilated rooms with foul air, than in the wards of a well-ordered hospital, and it is very difficult to resist the evidence of contagion afforded in some cases under the former circumstances. If contagious, the poison is at least much less volatile than those of the more strictly contagious diseases.

ETIOLOGY.

The last five years have brought us apparently but little nearer a definite solution of the origin of typhoid fever, or of the process of elaboration of the poison producing the disease; but the manner of its transmission to man has been further elucidated by the continued careful investigation, especially in England, of drinking water and milk supplies, and illustrated by an extensive epidemic among the frequenters of a musical festival at Kloten (Zürich), seventy per cent of whom were attacked with a disorder which numerous autopsies showed to be typhoid fever, in so far as an autopsy performed by skilful hands is still accepted as proof of this disease. Some of the meat consumed at this festival is reported to have been putrid, and the symptoms manifested by many cases would indicate the disturbing element of septic poisoning. The chief source of the poison in this outbreak, however, was traced by so competent an observer as *Huguenin* to veal coming from a sick calf. Persons living away from Kloten, who were not present at the festival, but who purchased and eat the brain and lungs of this animal, were similarly taken ill. Confirmation of *Huguenin's* unavoidable conclusion was believed to have been found by another observer, *Walder*, in the apparent communication to their own calves by some of those infected at Kloten of a disease producing intestinal and glandular lesions, resembling those of typhoid fever in man.

Curiously enough, an outbreak of sickness occurred at Andelfingen, in this same province of Zürich, in 1839, among five hundred persons who partook of *putrid* meat. This was long cited and generally accepted as proof of an autochthonous origin of typhoid fever. The meat was simply putrid, it was both veal and pork; the animals from which it came were not known to have been ill, and it has been since shown (*Liebermeister*) that the disease was not typhoid.

The Kloten outbreak stands on a different basis, and the number and character of the physicians who attended those attacked, assisted at autopsies, or report the circumstances, lend additional value to the occurrence.

Every year our knowledge of the intimate nexus which binds together our domestic animals and man seems to become closer, and we are slowly becoming aware that the presence of such diseases in animals is fraught with evil consequences to ourselves. Of late we have begun to suspect that typhoid fever and diphtheria may be in some cases communicated to the human subject, not merely by milk, but through the cow by some modified disease which we do not know (*Greenfield*).

In the contamination of milk supplies, whether by a legitimate use of infected water for washing dairy utensils, by its illegitimate use for dilution, by cattle themselves diseased, or bringing on their bodies poisonous particles from fields manured with the excrement of human beings suffering from typhoid fever; and again in diseased meat we have two modes of transmission which were hardly thought of until quite recently, and in contaminated water-supplies a third mode, which, though generally accepted, was not admitted by so distinguished a sanitarian as *v. Pettenkofer* four or five years ago. Other now unsuspected modes of transmission will undoubtedly be traced in the near future. In the mean time, the source of many outbreaks is clear which would before have remained a mystery, and the temptation is diminished to resort to so unsatisfactory a theory as the pythogenic for a source, or to so unphilosophical a belief as that in spontaneous or equivocal generation to explain what we cannot understand in the transmission of this disease. Epidemic outbreaks and sporadic cases, inexplicable under our present knowledge and methods of observation, are and will be reported.

Typical of such epidemics, though longer than most in duration, was that occurring at Ascot, which prevailed intermittently during a period of four and a half years. It was worked up by Dr. *Ballard*, a competent and skilful investigator. There were two apparently distinct outbreaks, the first apparently ending in September, 1875, the other apparently commencing in July, 1876. Before the first outbreak, no similar epidemic of enteric fever had been known at Ascot for over forty years. The inquiry left no doubt as to the relations of the milk of a particular dairy-farm with the epidemic; it was found that of 68 cases of fever in 39 families, no less than 58 cases in 31 families occurred amongst persons using this milk. Dr. *Ballard* found abundant opportunities at the farm for the contamination of the milk with filth, and especially with excremental filth, but was unsuccessful in establishing its contamination with a specific contagium. Not being content with the explanation of a *de novo* origin, and having to find an intermittent cause to account for the intermittent outbreaks, Dr. *B.* suggests several ways possible or probable for introduction of the specific poison. A theory based upon the geological formation of the country, and the conveyance of the specific poison in the subsoil water as a vehicle, down about half a mile of hillside, is the one preferred by him. Bearing in mind the epidemic at Lauten (*Liebermeister*), such a hypothesis is worthy of consideration, but it is precisely in connection with a milk epidemic like this that we may hope for more light in the future. In this instance, there is no report of the condition of the cows.

As an example of the sporadic class of cases, the following reported by a medical officer of health for an English rural sanitary district will serve:

Three members of a family of four were attacked successively in as many weeks with typhoid fever. They occupied a house consisting of four fair-sized rooms. It was built on a hillside, facing the junction of four wide valleys, "a more exposed situation could hardly be imagined." Above the house were miles of moorland; both house and privy were at a distance from any highroad, and quite out of the way of tramps. "There was an entire" absence of enteric fever from this district, a very thinly populated one; neither the father, the first victim, nor any of the others had been out of the district for a very long time, they had no visitors, the water supply was examined and found pure, the house was clean; the privy was full of filth and running over, and the fourth member of the family, a boy, who was not ill, is said not to have used this privy.

Given these data, and they are almost more than typical of those presented by the mountain farm house in Virginia, cited by the President of the American Public Health Association, of cases in New England reported by *Nathan Smith* sixty years ago, by Dr. *Stone*, of Maine, recently, and by many country practitioners who are shrewd observers, and we have, if we choose, a *de novo* generation of typhoid fever with its origin in filth. Such was the conclusion of the health officer, who was probably trained under the supervision of *John Simon*. The imagination, however, is no more exercised by the dried particle carried on the wind, and real knowledge is much furthered by a continuance of careful observations unbiased by preconceived theories until another such a step forward is taken as was made in tracing the milk epidemics, the first stage to which was the now universally accepted infection of water supplies; the Kloten epidemic again may present us with another link in this chain.

The advance in knowledge of the causes of disease by statistics is of a mathematical necessity extremely slow, as well stated by Dr. *Billings* in the introduction to Hygiene and Public Health of this series, and advance by the other most valued method, experiment and investigations in comparative pathology, is limited, as was there pointed out, by the fact that specific diseases seem usually specific to certain animals, and that many of the diseases of man cannot be communicated to the lower animals. The difficulties in this direction are increased in investigating typhoid fever by our continued ignorance of the specific germ or particle, the unknown factor of the problem. The last five years, however, offer encouraging advances in neighboring fields of inquiry. Dr. *Cossur Ewart*, following in the steps of *Cohn* and *Koch*, has shown by his investigations the close resemblance existing between the harmless *bacillus subtilis* and the dangerous *bacillus anthracis* of splenic fever; the existence of the *spirochæte obermeieri* in the blood of those suffering from relapsing fever has been pretty well established, the infectiveness of the blood has been proved by the inoculation of monkeys with the disease by *Carter* and *Koch*, and of men by *Motschutkoffsky*, the microphyte has been cultivated outside the human body by *Carter* and *Koch*, and the history of the organism worked out by several observers; the cause of an infectious disease of hogs, the hog plague or typhoid fever of the pig, a disease named by him "infectious pneumo-enteritis," has been announced by Dr. *Klein* to be a microbion very similar to the *bacillus subtilis*, which he has succeeded in cultivating, thus observing the various stages of its development, and in transmitting the same disease to mice and rabbits by inoculation; *Klebs* and *Crudeli* believe they have found the vegetable organism giving rise to malaria in man, and have produced, as they report, intermittent in animals by inoculation. M. *Pasteur*, pursuing the observations of Messrs. *Moritz*, *Perroncito*, and *Toussaint*, veterinary surgeons, considers it proved that a diarrhœal disease of chickens, "choléra des poules," is caused by a microscopic organism. This microbion is extraordinarily virulent, and M. *Pasteur* has not only cultivated it outside the living body, and reproduced the malady by inoculation, but shown that the disease is self-protective, and moreover that a modified microbion developed in the body of the guinea-pig occupies the same relation to this disease as the vaccine virus to small-pox.

Following in this general direction, *L. Letzerich* believes he has transmitted typhoid fever from man to rabbits by introducing by the mouth as well as by injecting subcutaneously inferior organisms (which he classifies as schistomycetes), suspended in distilled water, and obtained by repeated washings from the stools of persons affected with typhoid fever. In view of the preceding uncertain results of *Birch-Hirschfeld's*, and negative results of *Bahrdt's* experiments in this direction, these results of *Letzerich* stand greatly in need of further confirmation. According to this experimenter, the four rabbits receiving the injections died after a varying period of pyrexia, and a post-mortem examination disclosed infiltration of Peyer's patches, enlargement of the spleen, and invasion of the various organs and tissues, especially of the spleen and mucous coat of the intestines, by these schistomycetes.

Septic poisoning may possibly, in the opinion of some, account for all the changes observed; we will only venture to say that the experiments as well as the conclusions require much corroboration before we can regard typhoid fever as a "schistomycosis." They are mentioned merely as indicating a line of inquiry which has proved fruitful in connection with other diseases, and may yet disclose the unknown term in the typhoid fever problem.

In the mean time, we are doomed to constant disappointment in seeking in any one agent an exclusive or even controlling influence upon the propagation of this disease. It is always much more difficult to explain the existence and absorption of immense quantities of filth without typhoid fever, than the comparatively few cases of typhoid with filth surroundings, where the source of infection cannot be traced.

"Typhoid fever is by no means a disease of the filthiest towns, or of the filthiest parts of towns. Since I commenced investigating this question, I have been amazed at the immense amounts of urine and excrement—oxidized, incompletely oxidized, and as they come from the bladder and intestines—that are consumed in drinking water, and inhaled at the rate of 9,000 litres of contaminated air a day, and this for years, by young, old, and middle-aged persons, without any disease resulting that may be attributed to filth. I do not mean to deny the danger of filth. Especially filth decomposing without the free access of air constitutes, in my mind, one of the most important secondary factors of disease; one, too, which may become so essential a factor in some diseases as to absolutely be the one without which, in an aggravated form, the disease cannot exist, as, for example, in cholera, plague, and yellow fever" (*Folsom*). Dr. *J. Ewart*, after giving in detail a description of the utterly indescribable filthiness of the back-slums of many parts of the European and most parts of the native quarter of Calcutta, observes: "That the difficulty does not consist in not finding plenty of animal filth to account for any amount of enteric fever, but in understanding how it is not still more prevalent than it is in such an unsavory city. The 'reason why this form of fever is not more frequently met with amid such an abundance of fæcal matter in a state of putrescent fermentation, is probably to be discovered in the fact that fortunately most of it is not pent up in ill-ventilated sewers, but exposed daily to the powerful antiseptic influences of the atmosphere and sunlight in open drains, open privies and latrines, and in comparatively open cesspools. The impression which has been gaining ground of late years that, as the sewering of the town has been advanced, so has enteric fever been more frequently observed, lends countenance in support of this view. This augmented prevalence, however, may, in some measure, be owing simply to greater recognition of the fever, although it must not be forgotten that attention has been directed to the endemic existence of the disease during the past fifteen or twenty years.' "

Foreign physicians who have written upon the sewage, drainage, and sanitary condition of many different towns in China, including Pekin the capital, comment without exception upon the wretched condition of drains where there are any, the offensive methods for discharging the functions of drains where there are none, and the general neglect of sanitary laws in public streets and private houses. In this connection these same authorities call attention to the remarkable infrequency of enteric fever. In Canton only two cases of typhoid were observed by one practitioner during a period of more than ten years. Two physicians report an entire absence of typhoid and typhus from Amoy, though full of typical fever-dens. In Foochow only seven or eight cases of typhoid were seen by a Scotch physician during eleven years' practice, until 1872–'73, when four cases occurred. There seems to be the same immunity also from the other exanthemata, except small-pox: intermittent and remittent fevers are common. To the objection that much typhoid probably exists in such localities which is overlooked, it is fair to reply that it evidently does not occur with the frequency or severity which might be anticipated.

It is unnecessary to pursue this point further: filth does not create typhoid, though frequently affecting its development. Something is necessary besides the decomposition of healthy or even of albuminous (*King*) excrements.

Neither is the key to the whole subject to be found in low ground-water. The labors of *v. Pettenkofer, Buhl, Wagus, Buchanan* show that for some places, Munich, Berlin, etc., a periodic connection, at least, exists between falling ground-water and increasing typhoid, but the connection is not invariable for every year and for all places. Wet seaso as well as dry, are followed by and coincide with abundant typhoid. T inhabitants of the ground floor and lower stories of houses, in Berlin least, do not yield the largest ratio of typhoid cases. Of 1,087 cases typhoid fever recently investigated in Berlin, the results of which we published by Dr. *Skrezcka*, the ratio of the sick per 1,000 inhabitan increased from 0.83 in the basement to 0.84 on the ground floor, 0. and 0.95 in the first and second stories, 1.1 in the third story, and 1. in the fourth and fifth stories. Similar statistics are given for the 9 deaths from typhoid fever reported in the year 1875, excluding tho where the story of the house was not stated in the death returns; t figures are as follows: 0.89, 0.91, 0.95, 0.90, 1.03, 1.74. The calcul tions in both cases were based on the census of 1871; they may not absolutely exact, but are pretty nearly so, and relatively are quite The highest rooms being the cheapest, are occupied by the poorest, th least cleanly, the most crowded population. Other influences besides te luric thus make their weight felt. v. *Rothmund* states that in Aug burg considerable increase in typhoid occurs with scarcely noticeab changes in the level of the subsoil water. *Thomson* declares in regard t Melbourne, Australia, and the neighboring country that, from clinic histories, combined with all available telluric and meteorologic data, *Pettenkofer's* subsoil theory of typhoid fever malaria is found inapplicable. Drs. *Lewis* and *Cunningham*, in a report to government on soi changes as affecting disease in Calcutta, say: the greatest prevalence o fevers during the period of observation occurred coincidently with th period of highest water level.

Moreover, of late years the Munich observers themselves seem to hav desisted, for the present at least, from explaining the connection betwee fluctuations in the subsoil water and the frequency of typhoid, and t confine themselves simply to the continued repetition of the fact (*Liebermeister*, 1876).

Touching the influence of the season of the year upon the frequenc of typhoid, it may be remarked that, as far as observations go, the northern part of the United States, and certainly New England, offer no exception to the general rule that the frequency of typhoid increases as th summer advances; that is, typhoid increases in frequency from July t October or November, and then declines. Curves of fluctuation fo Basle, London, or Berlin would probably represent roughly most of ou towns. It is different where peculiar local conditions operate, as i Munich.

A statement of the deaths from typhoid occurring in Basle durin fifty years gives the following for the different months:

Jan.	Feb.	March.	April.	May.	June.
192	143	137	121	160	169
July.	**Aug.**	**Sept.**	**Oct.**	**Nov.**	**Dec.**
186	202	237	237	236	193

(*Hagenbach*).

Relatively this might stand for Boston, New York, or Chicago.

INFLUENCE OF AGE.—The same authority gives a table of the deaths from typhoid during 50 years in Basle according to the age so far as that was noted in individual cases, to which we have added the percentages. Total number of cases 2,059.

AGE IN YEARS.	NO. OF CASES.	PER CENT OF TOTAL.	DISPOSITION IN COMPARISON WITH AVERAGE =1.	AGE IN YEARS.	NO. OF CASES.	PER CENT OF TOTAL.	DISPOSITION IN COMPARISON WITH AVERAGE =1.
0- 5	108	5.24	.95	46-50	107	5.14	.94
6-10	85	4.12	.74	51-55	100	4.85	.87
11-15	106	5.14	.93	56-60	60	2.91	.53
16-20	239	11.12	2.03	61-65	53	2.57	.47
21-25	404	19.62	3.54	66-70	45	2.13	.39
26-30	290	14.08	2.54	71-75	26	1.26	.23
31-35	186	9.03	1.63	76-80	8	0.39	.07
36-40	143	6.94	1.25	81-85	2	0·09	
41-45	129	6.26	1.13	86-90	2	0.09	

SEWERS, DRAINS, WATER TRAPS AND CLOSETS.—Prof. *Frankland's* experiments with lithium in the sewers of London show that where the current is continuous and the liquid quiet, the lithium cannot be detected by spectroscopic examination in the sewer gas, but when the liquid is splashed and agitated it readily can be; for lithium substitute specific particles. It is well when possible to have sewers so arranged that they can be occasionally flushed with pure water. The importance of placing house drains in sight, of thoroughly ventilating them, of connecting with the sewer by iron pipe and hanging it to the house, are now better appreciated.

Dr. Carmichael, of Glasgow, has lately published some valuable experiments in regard to water traps and closets. He concludes that: Water traps are for the purpose for which they are employed, that is, for the exclusion from houses of injurious substances contained in the soil pipe, perfectly trustworthy. They exclude the soil pipe atmosphere to such an extent that what escapes through the water is so little in amount, and so purified by filtration, as to be perfectly harmless; and they exclude entirely all germs and particles, including, without doubt, the specific germs or contagia of disease, which, we have already seen, are, so far as known, distinctly particulate. Other sources of contamination must be looked for, except where there is some faulty construction of the trap itself, and these faults of construction are sufficiently numerous. Among other sources of foul air than faulty traps he considers none more common and pestilential than the ordinary pan water closets, with its perpetually nasty trunk. His views in regard to the pan water closet are those of *Ford*, of Philadelphia, expressed in *Buck's* Hygiene, of *Bird*, of *Latham*, and it is to be hoped of all intelligent people who unfortunately know anything about it.

CASES WHICH RUN AN IRREGULAR COURSE.—Recent years have scarcely put us in a more favorable position for discussing this part of our subject with profit. Materials and observations have accumulated, especially in the United States, but the difficulty of differentiating a diagnosis between mild typhoid and "cess-pool" fever without an autopsy still exists, and the pathological distinctions between the lesions caused by typhoid fever and septic poisoning are not sufficiently defined to make an autopsy in all cases a positive indication. These fundamental difficulties are reflected in the views of good authorities and of practitioners of extensive experience. The utmost caution is still required in judging of such cases.

The influence of malaria in excluding or modifying typhoid is also an important factor.

Prof. *Clark*, of New York, thinks the question is a little unsettled whether the excretions from healthy persons can produce typhoid fever; his impression, however, is that, when these cases are closely examined in the new light, they will be found to be what is described as "cess-pool fever." Dr. *Loomis* ascribes the adynamia in the so-called typho-malarial fevers to pythogenic influences, which cannot, he thinks, cause enteric fever in the absence of a specific germ. Dr. *Cabell* reports that this is the view taken by many Southern physicians, with whom he has corresponded on this subject, and whose residence in malarious regions gives them opportunities for extensive observation. The majority of his correspondents express the common opinion that typhoid and malarial fevers have each its distinct specific cause, and both may prevail at the same time in the same place; a small proportion of these believe that the two diseases may co-exist in the same individual as a hybrid typho-malarial fever. This is the view advocated by Dr. *Woodward*, supported by Dr. *Hoff*, and accepted by Dr. *Cabell* himself. This last gentleman, however, acknowledges, as we believe very properly, a certain degree of antagonism between typhoid and malarial poisons, that enteric fever is far less common in malarious regions than elsewhere, and that its frequency increases in these very regions during periods when there is a suspension of malarious influence.

Dr. *Hoff*, surgeon in the U. S. Army, gives the clinical histories of five cases of a disease which he identifies with *Woodward's* typho-malarial fever, which occurred among troops under his charge in Wyoming Territory, not far from the Rocky Mountains. He considers himself forced to attribute the source of the poison to the water in a stream having its origin in the melting snow of the mountains. The cases were all of the same type; there was one death in which an autopsy revealed the characteristic lesions of typhoid fever. Dr. *Hoff* sums up his observations and conclusions in brief as follows: 1st. The fever of the Rocky Mountain region is a hybrid disease, the prominent features of which are typhoid, the modifying, intermittent. 2d. It appears during or after exposure to field service, generally, though not necessarily, in late summer and early autumn, and seems to bear no relation to typhoid infection as now usually accepted by the profession. 3d. At its inception, this disease manifests itself as an intermittent of quotidian, tertian, or other form; this stage is followed, in about two weeks, by the typhoid stage, lasting about four weeks, in which typical typhoid symptoms may be observed, modified in a greater or lesser degree by intermittent indications. 4th. The pathological anatomy of the disease is that of typhoid fever. 5th. The treatment should be antiperiodic and antipyretic.

TREATMENT.

Next in importance to the etiology, as measured by the amount of attention it has received of late years, comes the treatment of typhoid fever.

In reference to the general treatment of the disease and to especial manifestations, Sir *Wm. Jenner's* address delivered in Birmingham, before the Midland Medical Society, forms, on account of that distinguished gentleman's long experience and generally recognized authority upon this subject, a very valuable contribution. He believes that, in the present state of pathological knowledge, it is impossible to fix the treatment of typhoid fever on a more sure basis than individual opinions founded on experience. Owing to the complexity of the disease and the great individual differences in the sick, he regards it as scarcely possible to find two cases in all respects identical, and quite impossible to collect records of a sufficient number of cases practically identical to determine by numerical analysis the best mode of treatment. This leads him to advocate careful attention to the symptoms in each case as they arise, and so-called specifics either as antidotes or forms of treatment do not find favor in his eyes. The fever he would meet by rest, quiet, fresh air, mixed liquid food, and bland diluents, and the exclusion of fresh doses of poison; the intestinal lesion by the careful exclusion from the diet of all hard and irritating substances, and the removal from the bowel of any local irritant. Of pure water internally, *Jenner*, with *Liebermeister*, advises in this disease an unlimited supply, and this advice is worth taking to heart.

DIET.—In common with others, *Jenner* admits the value of milk as an article of diet in fever, but cautions against its indiscriminate employment in almost unlimited quantities, reminding us that a pint of milk contains as much solid matter as a full-sized mutton-chop. Undigested and irritating curds, and very abundant fecal accumulations are the frequent accompaniments of a simple milk diet as carried to an extreme of late years not unusual. When catarrhal inflammation of the intestines is present, milk is the best diet; if the curd appears in the stools, the milk should be diluted with water or lime-water; when the bowels are torpid, beef or mutton broth may be given alternately with the milk, though neither of these is anything like as nutritious as milk (*Pepper*).

Opium, Jenner is convinced, is on the whole a most dangerous remedy. In the early stages of fever, it disturbs digestion and checks secretion; in the later stages of the disease, its influence on brain, heart, and secreting organs is sometimes fatal. With hemorrhage its use in enemas may be, and with perforation its use in some way would be unavoidable.

Calomel.—Dr. *Alonzo Clark* says we have been very familiar with the use of *calomel* in the treatment of typhoid fever in the United States, but that it has been pretty generally abandoned. Nor would a more general reliance be placed, as far as we can learn, upon any specific action to be expected from iodine either in altering the character or shortening the duration of the disease, whether administered as Lugol's solution or as iodide of potassium.

Alcohol.—When, forty years ago, Dr. *Todd* introduced to the profession the alcoholic treatment of fevers, he was as much alone as are those who now propose to do away with it entirely. Since that day the spectacle has been presented of the constant and indiscriminate use of alcohol in all forms of disease. To such an extent has this been carried that a large body of distinguished practitioners in England have felt called upon to issue a circular denouncing this indiscriminate use, and demanding a much greater restriction in its application (*Caswell*). The tendency at the present time in the United States and in Great Britain is undoubtedly a reaction against the excessive use, and toward a more rational use of alcohol in fever, and especially in typhoid. Stimulants are only demanded for the relief of certain symptoms (*Pepper*). Alcohol, by its influence on the nervous system, is of the greatest value in the treatment of typhoid fever, but it should only be given for the purpose of attaining a definite object; its effects should be watched that the desired result may be obtained from as small a dose as possible. It calls forth strength, but does not create it, and is therefore more frequently indicated and in larger doses in the later stages.

Tremor out of all proportion to other signs of nervous prostration is evidence of deep destruction of the intestine. A small, deep slough will be accompanied by great tremor; a large extent of superficial ulceration may be unattended by symptoms. Now it is deep ulcers following separation of deep sloughs which are especially liable to give rise to severe hemorrhage and perforation. In these cases of tremor, alcohol should always be given to increase nerve energy and to limit sloughing and ulceration. Delirium is, as a rule, one of the symptoms which are influenced for good by alcohol, but in which its effects should be most closely watched. When the urine contains a considerable quantity of albumen, alcohol should not be prescribed, unless for the relief of some symptom immediately threatening life, and then with the greatest caution. It is in exceptional cases only that more than twelve ounces of brandy in the twenty-four hours can be taken without inducing some of the worst symptoms of prostration. Nearly all the good effects of brandy, when its use is indicated, are obtained by four, six, or eight ounces in twenty-four hours. When in doubt of the wisdom of giving alcohol in a case of typhoid fever I do not give it, and when there is a question of a larger or smaller dose I prescribe, as a rule, the smaller (*Jenner*). The use of stimulants is regulated by the state of the pulse; if the beats are growing in rapidity and losing in strength, if they pass 110, 115, 120, a half-ounce of spirits or a glass of champagne is given; if there be from any cause doubt of the need of alcohol in a rising pulse the dose is given with the finger on the wrist; if the pulse steadies or slows, the wine is repeated on its rising. Twelve ounces of spirits daily is said to be all that the worst case needs, but this limit is constantly and necessarily exceeded with favorable results (*Stedman*).

ANTIPYRETIC TREATMENT.—The combat with pyrexia has gone on briskly, and quinine, salicylic acid, salicylate of soda, digitalis, alcohol, and cold baths continue to be experimented with as antipyretics, whether separately in larger or smaller doses, or in varied combinations. There is nowhere great unanimity of opinion as to the positive value of these agents, or the best methods of administration, but the evil effects of excessive or prolonged elevation of temperature are generally conceded, as well as the fact that, in some uncomplicated cases, an otherwise inevitably fatal result may be averted by the timely employment of one or more of these antipyretics.

In general, it may be said that, in Germany, where professional attention was recalled to the importance of controlling febrile action in typhoid, the antipyretics still retain the confidence originally expressed in them, though in a modified degree; in France, they have gained a place, though their use there has certainly never excited much enthusiasm; perhaps the German indorsement proved unfavorable to them; whilst in the United States and Great Britain, where the experiments with cold baths at least have been much less extensive than in Germany, there seems little disposition to adopt cold baths as a constant form of treatment, though their value is recognized in certain cases and at certain periods of this disease. Many good authorities in those countries doubt the wisdom of the employment of large doses of quinine and salicylic acid, whilst others seem more than satisfied with the results obtained from these drugs. The truth is, that some patients bear best one form of antipyretic and others another, and, in a certain proportion of cases, all fail, either partially or wholly, to effect the object aimed at. If, in any given case of typhoid fever, the pyrexia gives cause for anxiety, and fifteen or thirty-grain doses of quinine are well borne, without giving rise to symptoms of nervous or gastric irritation, the administration of these full doses, especially at evening, will often control the fever. Salicylic acid is now less used than salicylate of soda; salicin is thought to produce a more prolonged, though slower effect upon the temperature than quinine; and in combination with this last the others are considered to increase its efficiency and to diminish the liability to relapses, which most authorities are now agreed is greater under antipyretic treatment. When any or all of these drugs are ill borne or insufficient, resort must be had to the application of cold—whether in the form of cold bath, gradually reduced bath, sponging, or sprinkling on a sheet—and that either alone or in conjunction with the above drugs. There is no doubt that, when quinine is well borne, its combined use diminishes the number of baths required.

Each case must be its own rule for the dose of the drug, for the form of application of cold, and if baths are given, for the temperature and duration of the bath. Mild cases do not require baths, and, in advanced cases (the third week) they are not safe. Great care should undoubtedly be exercised in administering them to young children and old persons.

Liebermeister, in Germany, and *Dumontpallier*, in France, now employ an apparatus for applying cold. There is a refrigerating envelope through which the water runs, and by means of stop-cocks and thermometers the temperature of the body can be lowered continuously or intermittently, and the change effected readily noted.

In a late reply to a vigorous attack by Prof. *Gairdner*, of Glasgow, upon the German antipyretic treatment, *Liebermeister* says: The antipyretic treatment is not, as you believe, a matter of routine. On the contrary, with it the cases are more individualized than with any other method of treatment. Each agent is used only when necessary, and when none is necessary none is used. But, in order to know what is necessary, one must, of course, observe each individual patient much more thoroughly than has hitherto been customary. It is just the routine hitherto pursued for which we propose to substitute a method of close observation, very exacting on the medical attendant, which offers the greatest obstacle to the introduction of the antipyretic treatment.

Dr. *Austin Flint* says that his observations as regards the antipyretic treatment are sufficient to lead him to believe the statement by *Liebermeister* that, under this treatment, "the old picture of a typhoid fever patient is no longer to be seen," to have a solid foundation in clinical experience, although somewhat extravagant, and to concur with this writer in saying that thereby "typhoid fever has lost a great part of its terrors."

Drs. *Edes* and *Stedman* report the use of graduated baths, combined with internal antipyretic treatment, in a considerable number of cases, at the Boston City Hospital, with favorable results.

Out of 32 cases entering, during the first week of fever, in Dr. *Edes'* service, in the course of three years, 1872–1874, there was only one death. A series of 46 cases entering this hospital at an earlier period, likewise during the first week, but not treated antipyretically, showed a mortality of a little over 10 per cent.

The statistics of the results of treatment and percentage of mortality at the Bellevue Hospital, New York, since the introduction of antipyretics, up to the year 1879, are not of a character to be of much practical value, but, as far as they go, are not especially favorable. Graduated baths have been tried there a number of times, and the opinion is expressed that they are uniformly annoying and depressing to the patients. At the Massachusetts General Hospital, antipyretics, and certainly cold baths, have not been systematically tested, but the usual rate of mortality at that institution, about thirteen per cent, is not an unfavorable one. Dr. *Smythe*, of Indiana, from an experience of eighteen cases, considers cold baths and quinine as indispensable in typhoid, but the latter as the more valuable of the two.

Dr. *Nathan Smith* published a short essay on Typhoid Fever in 1824, in which there would be little to change, even at this date; in it he gives the results of his experience of this disease in New England, and shows that he was in the habit, since the latter part of the last century, of using cold water, when indicated, externally as well as internally, with freedom and courage, and that he recognized its value in certain cases. Sir *Wm. Jenner* says that neither his own limited experience nor the evidence adduced by others in its favor has carried conviction to his mind of the advantage of the treatment by cold baths. At the same time, he recognizes the fact that the direct cooling of the body is, in some cases, essential to the preservation of the life of the patient, and mentions with favor the graduated bath, tepid sponging, the wet pack, and cold to the head by the india-rubber tubing cap. He expresses himself as having been disappointed in the effects of quinia and salicylate of soda as reducers of temperature, while he has occasionally seen both do much harm by disturbing the stomach and interfering with digestion. Dr. *Pepper* expresses himself even more strongly, and says that he never administers the enormous doses of quinia given by German physicians; that there is so much unnecessary irritation of the mucous membrane produced that heroic treatment, such as this, should only be adopted as a last resource; that he is in the habit of giving about twelve grains in the twenty-four hours.

The graduated bath was adopted at St. Thomas' Hospital, London, and Dr. *Ord* says, in regard to its use there, which was quite general, that the practice, though not uniformly successful, has not, in any instance, done any known harm, and has, in all probability, averted death in several cases.

In regard to the graduated bath, Dr. *Greig Smith*, of the Bristol Royal Infirmary, is confident (though he thinks the contrary is very generally believed) that the endeavor to prepare the system for a temperature of 60° by lowering it from one of 90° in half an hour or so causes more depression than to plunge the patient at once into the requisite cold. It certainly causes more discomfort. In this opinion Dr. *Collie*, of the Homerton Fever Hospital, where baths have been successfully used, coincides. Moreover, he says they have never given more than three baths in twenty-four hours, and should not, in any case, give more than four; that the duration of the bath should not, as a rule, exceed ten minutes, and, in children of ten, not more than seven minutes; that, in most cases, the bath should be discontinued toward the end of the second week, and, if an antipyretic is needed, quinine should then be given. To young children and elderly persons quinine should be given instead of the cold bath.

Mr. *Murphy*, Resident Medical Officer of the London Fever Hospital, writing in 1877, stated that up to that time the cases of typhoid treated antipyretically in that institution had been too few to urge as statistical proof of the efficacy of that treatment, but the results on individual cases had been sufficiently satisfactory to lead to its general adoption in this hospital. He further says: It is not contended that it will insure the recovery of every patient suffering from enteric fever; that it will save life in those patients who die in the second week of their illness from the direct influence of the poison; nor that, when commenced late in the third week, it will undo the injury that has already occurred. It is not believed that it will shorten the period of illness, for it has even appeared to prolong it; although this may be due to the fact that the more severe cases have been bathed—cases which would under any circumstances have run a long course. But there is a large class of cases which, under the expectant treatment, die at the end of three or four weeks, worn out by the continued pyrexia, and these can without doubt be saved by an early and systematic antipyretic treatment. Without the bath, quinine will only control the temperature in the milder cases. Quinine will never altogether replace the bath, for it is not followed by the favorable effects of the latter on delirium and in producing sleep, but in conjunction with the bath it is a most useful aid; sponging or wrapping in sheets saturated with cold water are useful in mild cases, but cannot be relied on in severe ones. The difficulties of the treatment are inconsiderable, it is not attended with any danger, the patients rarely object to it, and by some it is liked. The reduction of delirium, the quiet sleep, the general feeling of comfort it gives the patient, are sufficient to convince the most sceptical that the cold bath is one of the most useful therapeutic agents we have in the treatment of enteric fever.

The following from the *Revue des Sciences Médicales*, 1878, reflects very fairly the French estimate of baths up to that period. To our knowledge, there is not a single hospital service in France where baths are used as a general method of treatment. M. *Féréol*, one of the physicians who recently has resorted the most to baths as a method of treatment, cited a series of forty-three cases, of whom only two were bathed. M. *Bernheim*, at Nancy, says that often out of ten or twelve cases of typhoid only one will require baths. M. *Raynaud* regards baths as an energetic form of treatment, with which one should not be extravagant, but reserve it for cases where the situation of the patient is sufficiently grave to authorize the physician to combat it.

One must, moreover, distinguish the conditions in which baths should be ordered regularly and continuously, the only true refrigerant method, and those cases where occasionally in the course of the disease recourse is had in a temporary manner to some one of the processes for applying cold.

Physicians at Paris who have had recourse to the new method of baths agree that for grave cases the advantage is on the side of cold baths (*Féréol*); some are inclined to think that a number of recoveries would not have been procured by any other method, and think they cannot with certainty attribute any death to the treatment. It does not seem probable, however, that cold baths will be accepted in France in the immediate future as a general method of treatment in typhoid fever, but the indications for their use are constantly better defined, and the different processes of hydrotherapy are gradually more and more employed, whether as a regular and constant application in certain severe cases, or in a casual way, and as an exceptional measure to combat temporary complications. They are not a specific, but a very energetic agent destined to bestow great benefits in proportion as we understand better the conditions in which they are to be resorted to (*Homolle*).

M. *Peter*, in an article which is robbed of much of its value by a too evident anti-German feeling, attempts to show that the treatment by baths is less a process of cooling than one of revulsion; that its good effects do not arise from the lowering of the temperature, but from a profound perturbation of the nervous system; in short, that the lower temperature is but a very indirect result of the treatment. He complains that in the struggle of the "hypo" with the "hyper" of "cold" with "warmth," the patient has disappeared altogether. That there are no longer therapeutics or treatment of symptoms, but a mere arithmetical problem, a subtraction. He thinks, apparently with *Jenner*, that the best therapeutic system, especially in the treatment of typhoid fever, is, and always will be, to have none.

Raynaud's experience with the treatment by cold baths in the Lariboisière Hospital was rather favorable than otherwise. In the years 1874–1876, after excluding the mild cases, there were 117 typhoid patients with 17 deaths, 2 of which occurred immediately after entrance; this gives a mortality of 14 per cent. During the year 1873, out of 30 cases under expectant treatment, there were 8 deaths, 26.7 per cent. He attaches more importance to prolonged pyrexia than to mere elevation; after the twelfth day of the disease he expects but little benefit from baths, and never applies them after the eighteenth day. Given a temperature of 104°, lasting five to six days with only slight remissions, he would regard the cold bath as a necessity. A bronchitis would not offer an impediment to baths, pneumonia and cardiac weakness with small pulse are decided counter-indications.

M. *Foltz* found physiologically that cold clysters have a local action on the intestine, and a general action shown by a decided slowing of the pulse and fall of temperature after a lavement of half a litre of water at 50° Fahr. This coincides with Dr. *Rutenberg's* experiments. In febrile conditions analogous effects are produced; moreover, nervous manifestations are soothed, thirst is appeased, the appetite is stimulated, the secretions augmented. These effects are the more manifest and persistent the lower the temperature of the water, the greater the quantity injected, and the more frequent the injections. M. *Foltz* orders, as a rule, lavements of about a pint at 50° to 55° Fahr., repeated every two, three, or four hours, or at longer intervals during sleep or when the temperature falls. In his cases, the number of lavements varies from thirty to three hundred for each case.

Twenty-seven typhoid patients were treated, with one death, fifteen of these had a severe form of typhoid. This mode of treatment has objections which readily present themselves, and is not likely to become general, but may be occasionally of service.

Prof. *Immermann* has published some interesting remarks on the prevention of relapses. Between 1872 and 1877, *Immermann* treated about 1,200 cases of typhoid fever in the hospital at Basle; 15.6 per cent of these suffered relapses. The proportion of relapses varied in different years from 12 per cent to 19 per cent. Having in vain guarded his patients from all error in diet, and kept them in bed until the end of the second week of continued absence of fever, he was unable to doubt that the relapses were independent of external influences, and in the great majority of cases arose from the liberation of unconsumed portions of the original poison. He attributes the increase in the proportion of relapses, from 8 per cent to 10 per cent ten or fifteen years since, to the present percentage, indirectly and directly to the new antipyretic treatment. Indirectly, on account of the diminished mortality; directly, on account of the use of hydrotherapy and antipyretic medication which interfere with the natural destruction of the poison in the course of the disease. To combat this result, he had recourse to a secondary disinfectant treatment. To twenty-two convalescents he administered, from the first day with absence of fever, daily doses of from sixty to ninety grains of salicylate of soda, and continued it during ten or twelve days. Of these twenty-two, only one had a relapse, and in this case there was a grave imprudence in diet. Among 93 patients not submitted to this supplementary course there were 27 relapses. A second series of experiments gave 1 relapse out of 29 convalescents of both sexes treated with salicylate of soda, and in this case the exhibition of the medicine was begun by mistake only on the fourth apyretic day; 67 other cases of typhoid offered 15 relapses. To sum up, *Immermann* experimented during two successive years, 1877, 1878, with salicylate of soda upon 51 convalescents from typhoid, among whom the relapses did not exceed 4 per cent, whilst amongst 160 others not so treated, the proportion of relapses amounted to 23.6 per cent.

The statistics of Dr. *Goltdammer* in regard to the treatment by cold baths, in the Bethanien Hospital in Berlin, are the most extensive which have appeared of late years. They cover a period of nine years, from 1868 to 1876 inclusive, during which 2,068 cases in all were treated, with a mortality of 13.2 per cent. They deal with a homogeneous material, subjected to similar general influences, are full, carefully compiled, not made to support a pet theory, and their value is enhanced from their being comparable, to a certain extent, with a series of 2,228 cases during the years 1848 to 1867 inclusive, drawn from the same population, subjected to similar general surroundings, and treated in the same hospital on the expectant plan. In making up the report, the former different use of the terms "typhus" and "febris gastrica" has been taken into acccount, and the rate of mortality in the early series thus rendered more, rather than less favorable. Moreover, no proof exists that the type of typhoid fever has changed in Berlin of late years. From the early series of 2,228 cases during the years 1848 to 1867, there were 405 deaths: 18.1 per cent. From the second series, 2,086 cases during the years 1868 to 1876, there were 267 deaths: 13.2 per cent. A diminution in mortality of 5 per cent in favor of the period of cold baths is shown, that is to say, 100 deaths were avoided during the period of cold-water treatment.

The average length of time in the hospital was:

1858–1867 incl. (for 1,086 cases)...............	46.1 days.
1868–1876 incl. (for 1,519 cases)...............	39.8 days.
Difference,	6.3 days.

There was thus a difference of 5 per cent in mortality, and of 6.3 days of treatment in favor of the period of cold baths. In the preceding series, all cases, the light, the severe, and the hopeless are included. In order to arrive as nearly as possible at an approximative mortality during the period of hydrotherapy, Dr. *Golldammer* deducts from the second series sixty-four deaths, occurring when the patient was brought in too late to use baths, or in a condition already hopeless from existing complications, and thus obtains a mortality of 10.5 per cent for the cases subjected to cold baths. The method he designates as a tolerably severe, but not excessive one. The baths were given at a temperature ranging from 68° to 82° Fahr., generally about 75°, with a duration of from ten to fifteen minutes. Sensitive patients, or those with cardiac weakness, were given baths of 88° Fahr., lowered in the course of fifteen or twenty minutes to 78°.

Temperatures were taken in the axilla every three, and in severe cases every two hours, and whenever 104° was reached a bath was given. Of the patients bathed regularly, the majority had three or four baths daily, the more severe cases five or six, and a very few seven. Baths were omitted in the night. All cases were subjected to treatment in which no counter-indications existed, or in which the course of the disease was too light to justify treatment. As counter-indications are mentioned especially cardiac weakness, also stenosis of the larynx, lobular pneumonia, pleurisy, intestinal hemorrhage, peritonitis. Salicylic acid and quinine were occasionally given in the evening in cases of persistent high temperature. It was not found that the baths were agreeable to the patients, but neither was there much resistance, and they were certainly quieter, slept better, and were less troubled with bed-sores. One case of syncope in the bath occurred, with subsequent fatal result. The records of the last three years, '74, '75, '76, being more full, present some interesting tables which are appended. An increase in the proportion of hemorrhages from the bowel was not observed.

The statistics of Dr. *Golldammer* give no positive information as to the relative frequency of relapse under the expectant and the cold-water treatment, as there were no exact data on this point in the early series of cases; his opinion, however, coincides with that of other observers, being that the proportion of relapses is increased by the baths. By excluding indiscretions in diet, and fresh infection from without, he concludes that in the great majority of cases the relapse had its origin, whatever might have been the exciting cause, in the same infection which caused the first attack. This view is also somewhat strengthened by the fact that most frequently the mild, short, abortive forms of typhoid, less often the moderately severe, and rarely the severe cases were those followed by relapse.

One case passed through a pretty severe typhoid in this hospital, returned ten weeks later with another severe attack, which was followed by a relapse. This individual, therefore, experienced three clearly marked attacks of typhoid within six months.

The apyretic interval preceding the relapses varied from 3 to 21 days, and averaged 8 to 9 days. Among complications and causes of death for these 783 cases were perforation 13 times; 51 times hemorrhage from the bowels = 6.5% with 21 deaths; 9 times diphtheria with 8 deaths and 7 unsuccessful operations; and 13 times pleurisy with 8 deaths.

MORTALITY ACCORDING TO AGE.

AGE.	NUMBER.	DEATHS.	PER CENT.
4-15 years	51	7	13.7
15-20 "	224	27	12.
21-25 "	274	42	15.3
26-30 "	121	21	17.3
31-40 "	73	18	24.6
41 years and over	32	15	47.
	775	130	16.7

MORTALITY ACCORDING TO PERIOD OF ADMISSION.

PERIOD OF ADMISSION.	NUMBER.	DEATHS.	PER CENT.
Unknown	33	..	..
1 week (1-4 days)	113	9	7.9
1 week (5-7 days)	215	23	10.7
2 weeks	335	67	20.
3 weeks and later	87	31	35.6
	783	130	16.6

The duration of fever in those who recovered before and after entrance into the hospital was:

For men	21.9 days.
For women	24 days.
	22.9 days.

RELAPSING FEVER.

BIBLIOGRAPHY.

Obermeier: Weitere Mittheilungen über Febris recurrens. Berlin. klin. Wochenschr., Nr. 35, 1873.—Budberg: Ueber Febris recurrens. Diss. Berlin, 1873.—Semon: Zur Recurrens Epidemie in Berlin, 1871-72. Diss. Berlin, 1873.—J. Simon: Relapsing Fever in the Metropolis. Lancet, I., p. 72, 1873.—F. Baemisch: Die Complicationen und Nachkrankheiten der in der Greifswalder Klinik behandelten Fälle von Typhus recurrens. Arch. f. klin. Med., XV., S. 53.—Litten: Die Recurrens-Epidemie in Breslau im Jahre 1872-73. Arch. f. klin. Med., XIII., S. 128, 1874.—Ponfick: Ueber das Vorkommen abnormer Zellen im Blute Recurrens-Kranker. Centr. Bl. f. med. Wiss., Nr. 25, 1874.—The same: Anatomische Studien über Typhus recurrens. Virchow's Arch., Bd. 60, S. 153.—Birch-Hirschfeld: Ueber die Spirillen im Blute Recurrenskranker. Arch. f. klin. Med., XIII., S. 346, 1874.—Münch: Protokolle der Sitzungen der Gesellschaft odessaer Aerzte, VII., 1874.—Unterberger: Febris recurrens im Kindesalter. Jahrbuch für Kinderheilkunde, N. F., X., S. 184, 1876.—Heidenreich: Ueber den Parasiten des Rückfallstyphus. Berlin, 1877.—H. V. Carter: Notes on the Spirillum Fever of Bombay. Med. Chir. Trans., LXI., p. 273, 1878.—Motschutkoffsky: Materialien zur Pathologie und Therapie des Rückfallstyphus. Arch. f. klin. Med., XXIV., S. 80, 192, 1879.—Holsti: Om rekurrensepidemin i Helsingfors under åren 1876-77, äfvensom Sannua Sjukdoms uppträdande i Finland. Nordiskt med. Arkiv, XI., 1, 1879, Stockholm.—F. Cohn: Zur weiteren Kenntniss des Febr. recurrens und der Spirochaeten. Deutsch. med. Wochenschr., V., S. 189, 1879.—Koch: Die Uebertragung der Recurrens-Spirochaeten auf Affen. Deutsch. med. Wochenschr., V., S. 327, 1879.—L. Riess: Weitere Beobachtungen über Febris recurrens. Deutsch. med. Wochenschr., V., S. 651 u. 663, 1879.—H. V. Carter: Contributions to the Experimental Pathology of Spirillum Fever, No. 1. Med. Times and Gaz., I., p. 304, 1880.—Enke: Ueber den Rückfallstyphus. Magdeburg, 1879.—Lewis: The Presence of Micro-organisms in Normal and Diseased Blood. 1879.—W. Friedreich: Das Auftreten der Febris recurrens in Süddeutschland. Deutsch. Archiv f. klin. Med., XXV., S. 518, 1880.

This disease is not indigenous in the United States. Cases were reported by *Clymer* in 1844, and by *Flint* in 1850–51. The malady was confined to recent Irish immigrants and did not spread from them. It was reimported again in 1869, and prevailed to some extent in that and the following years in New York and Philadelphia, where its diffusion was, according to *Flint*, undoubtedly due to a contagium. Since its disappearance in 1870 nothing more has been seen of it in this country. We have been unable to learn of any epidemics of infectious disease answering to the description of bilious typhoid or relapsing fever as having prevailed among the negroes of Philadelphia and North Carolina, and, therefore, cannot share the opinion expressed by Prof. *Hirsch*, that this disease was the cause of a peculiar mortality among those people.

The old term, famine fever, which is now discarded, indicates, not that the symptoms included under the name are caused by want, but that want, overcrowding, and their concomitants favor the development of the disease. The name, however, serves to remind us that the present distress in Ireland, the home of relapsing fever, is very liable to be followed by a general outbreak of this disease at various points, and increasing emigration would in all probability bring it again to our shores.

In 1873 *Obermeier* described a spirobacterium found by him in the blood of those attacked with relapsing fever. This was identified as the spirillum, or more properly the spirochæte of *Ehrenberg*. These two microbions form respectively the fifth and sixth group in *Cohn's* classification of schizomycetes or schizophytes, and compose Tribe IV., that of spirobacteria. As both names have been applied to the microphyte of relapsing fever, they will be used interchangeably in the present article, though spirochæte is the more correct designation. Since this discovery by *Obermeier*, much attention has been given to the etiology and pathology of relapsing fever, materials have been accumulated, and the relations of the microbion to the disease carefully investigated. It cannot be affirmed that positive results have as yet been reached, and many of the observations of those who have had opportunities to make such are very conflicting. This, however, is to be expected in the present transitional state of knowledge upon this and kindred subjects, where the best authorities are working in fields still new, and all are not equally experienced in methods of research.

Some investigators, as *Heidenreich, Vandyke Carter, Motschutkoffsky, Holsti, Enke,* believe they have found this parasite invariably, or almost so, at some stage in every case when the blood was examined microscopically; others, as *Riess* and *Lewis*, report a number of cases in which spirilla were not found during any stage of the disease, and again instances in which they were not discoverable during one or more periods of pyrexia. Such conflicting observations naturally make it desirable to suspend judgment as to the precise relation of the microphyte spirochæte to the disease relapsing fever. Whether it is simply attendant upon, or how far it is causative of the disease, we are not quite yet in a position to decide. It is a little premature to shout, "No relapsing fever without spirochætes, and no spirochætes without relapsing fever" (*Unterberger*). Our present knowledge would not justify us in declaring any given case as not one of relapsing fever because spirochætes are not found during the pyretic stage. When spirochætes are found in the blood, however, it is pretty safe to suppose that one has to do with this disease until it is shown, and that has not yet been done, that this parasite exists in the blood under other conditions. It is also necessary to remember that, as other hitherto unobserved agents may perhaps influence the febrile paroxysms, so other conditions of the blood besides temperature, such as amount of water, oxygen, etc., may affect the regular development of the microzyme spirochæte. The spiral bacteria found in various secretions and in water (*Billroth, Cohn, Ehrenberg*) are similar to our spirochæte, but careful inspection shows some differences, the spirochæte plicatilis found by *Ehrenberg* in water being somewhat larger, and, what is more reliable, inoculation does not produce specific results.

Both men and monkeys have been successfully inoculated with the spirillar blood of relapsing fever (*Motschutkoffsky*, *Carter*, *Koch*), and this inoculation carried to the second and third stages. Blood in which spirilla could not be found was successful in the hands of one experimenter, but it was taken in the earliest stage of a pyretic onset which soon developed the organism. Other non-spirillar blood did not produce specific results. There is no absolute proof that in these cases it was the spirochætes and not some other accompaniment of the injected blood which transmitted the disease. *Koch* announces that he has found this microphyte in the tissues of an inoculated monkey killed at the height of a relapse, something not previously observed and which we have not seen confirmed, and also that he has successfully cultivated it outside the living body, following the same methods which he pursued with bacillus anthracis of charbon; this had not been done previously except by *Carter*.

The further step, adopted in charbon and which forms the missing link, of reproducing the specific disease from the spores of the microphyte and from the organisms obtained from these, as far as we can learn, has not yet been taken. If *Cohn's* view is correct, that only bacilli form spores, the terms, though not the method, of the step would be altered. Even the successful attainment of this step would not be proof that no other agent caused relapsing fever. There are, however, limits to a rational and healthy skepticism. One thing appears from recent observations, that the spirochæte does not precede the pyretic attacks with sufficient constancy to be relied upon as a warning. To this short summary of the relations of this organism to the disease under consideration we add a brief synopsis of the more important recent investigations bearing on the subject.

All recent writers agree that one attack of relapsing fever does not protect the individual from a second entirely new one. *Litten* states that five fresh reinfections took place among the hospital patients at Breslau during the epidemic of 1872–73, whilst seventeen other patients who had already passed through the disease in 1868 were again attacked in 1872–73.

Dr. *J. Motschutkoffsky*, of Odessa, had a very favorable opportunity to study relapsing fever upon a rich material in the hospitals of that city between the years 1873–76; and he had the unusual advantage of being permitted to inoculate a number of human beings with the disease. During two years he made over two thousand preparations of the blood. In regard to the spirochæte, his observations led him to the following opinions and results: We do not yet know the origin nor the process of destruction of the spirochæte. It was never found in any of the secretions or excretions, and is not eliminated unchanged from the body. He reports having twice witnessed the process of its dissolution in blood into a finely granular detritus. *Litten* gives twenty-four hours, *Münch* over twenty-four hours as the length of time it retains its power of motion outside the body. *Heidenreich* saw it move one hundred and thirty days after being taken out of the body and sealed up in a glass tube, and thinks this organism preserves its activity best in a temperature of 58 to 70° Fahr. Our author saw it move thirty-seven days after being subjected to similar conditions. He was unsuccessful in cultivating it, as were also *Weigert*, *Lebert*, and *Heidenreich*, but *Koch* and *Carter* have since succeeded. The addition of equal parts of water to the blood is fatal to the spirochæte, but it also loses its activity more and more the thicker the blood becomes. Its activity is not affected by any internal exhibition of quinine, salicylate of soda, or other agents, and externally only affected by about 1¢ of quinine. It withstands a low temperature well, but begins to cease moving at anything below 32° Fahr.; it revives, however, in blood which is warmed after having been cooled to as low a point as 15° Fahr.

In regard to the relations of the microbion to the disease, this investigator thinks it impossible to deny the dependence in some way of the process of relapse upon the spirochæte; for he affirms that the appearance of a paroxysm and of spirochæte are nearly synchronous, that the termination of the paroxysm and the disappearance of the parasite are nearly synchronous, that inoculation with apyretic blood is unsuccessful, that occasionally the spirochæte may not be found in the first days of pyrexia, but that they must be present, as this blood is equally effective. During an attack spirochætes are a constant element in blood; during apyrexia, are found exceptionally.

He does not recall a single attack during his investigations, extending over three years, in which spirochætes were not to be found in the blood of the living. This agrees with *Heidenreich*. This latter alone on one occasion discovered this parasite in the blood of a person dying from relapsing fever, and then they were without motion. *Cohn*, *Ehrenberg*, and others have found organisms very similar to spirochæte in the secretions of the mouth, in water, in cysts, etc.; and *Cohn* is disposed to regard these different microphytes as representing merely so many different stages of the same. This is far from being proved. *M.* did not see spirochætes a single time, either during incubation or apyrexia, and noticed no difference in their size at different periods; in number they increase from a few hours after the beginning of the pyrexia up to about a day before the crisis.

He cannot accept *Heidenreich's* theory that the destruction of the microphyte is dependent on the pyrexia, as it does not agree with the facts, but thinks it more probably depends upon the degree of consistency of the blood. Though we cannot stop the disease by any remedies, we may, perhaps, be able to shorten the attacks by withdrawing liquid from the blood.

"Bilious Typhoid" of *Griesinger*, of which *Lebert* makes a separate division, and is rather disposed to regard as a distinct though similar disease in *v. Ziemssen's* 2d edition, 1876, *Motschutkoffsky* regards as unquestionably nothing more than relapsing fever with an hepatic complication, probably a catarrhal affection of the fine bile ducts. He bases this view upon the following observations: In sixteen cases of undoubted so-called bilious typhoid occurring in Odessa, he found the spirochæte in the blood in all, and the organism taken from the blood of the last eleven cases was subjected to the same tests as those taken from the blood of relapsing fever patients, with the same results. Secondly, an individual inoculated with the blood of a "bilious typhoid" patient developed, after the usual interval, the ordinary relapsing fever uncomplicated by icterus. Thirdly, a patient who was exposed to relapsing fever during convalescence from typhus, developed "bilious typhoid" with spirochætes in the blood; at this time there was no case of "bilious typhoid" in that wing of the hospital, nor had there been, as far as could be learnt by careful investigation, for more than two months; moreover, infection from without was rendered improbable by the length of time the patient had been in the hospital. *Holsti's* report of the epidemic at Helsingfors in 1876-77, in which the mortality was large, in which many cases exhibited marked icterus with other symptoms of bilious typhoid, though still running the usual course of relapsing fever, and showing spirochætes in the blood, is confirmatory of the Odessa observations, and of the identity of the two diseases. *Münch's* experience with autopsies upon those dying from "bilious typhoid" led him to call attention to the coincidence that the liver in many of these cases shows signs of previous pathological processes (cirrhosis, syphilis, etc.).

In the late Berlin epidemic (1879) *Riess* observed one case of "bilious typhoid" with plentiful spirochætes in the blood.

Motschutkoffsky sums up his experiments with the inoculation of spirillar blood briefly as follows: He did not succeed in inoculating animals, although monkeys were tried among others. Only blood was available. Milk, sweat, urine, saliva, and excrement were without effect. Blood was only effective during pyrexia; apyretic blood gave negative results. Blood taken during pyrexia, *e. g.*, the first hour of a commencing attack, was good, whether spirilla could be seen under the microscope or not. Artificial recurrens differs in no respect from the natural.

He did not find that reinoculation increased the virulence of the disease. Blood from recurrens biliosus produced simply recurrens. The period of incubation was from five to eight days. The period of apyrexia about equalled that of incubation.

In 1877, Dr. *H. Vandyke Carter* had an opportunity of observing a large number of cases of fevers in camp and in hospitals in Bombay. Between April and December, about three hundred and fifty cases presented themselves offering all the characteristics of the relapsing fever of Europe; in all these cases, spirilla were found in the blood, and *Carter* has applied the name "spirillum fever" to the disease. The questions raised by *Carter* of the influence of a malarial taint upon this disease are of interest, and will doubtless receive more attention. He regards want, crowding, and contagion as the immediate causes of spirillum fever. He examined the spirilla carefully, and his results correspond with those obtained in Odessa. He was unable to find spirilla except in the blood; he doubts the identity of similar filaments found in the saliva of fever patients, for they were present during the apyretic interval, and the same were found in his own saliva during health. He observed that the parasite has a natural tendency to seek a liquid medium, and he concludes that the relationship obtaining between the abundance of the parasite and the intensity of the fever is a contingent one, though commonly at particular times the connection between pyrexia and the parasite is close. He did not succeed in cultivating the spirillum outside the human body. Within the last year, 1879, and since the above investigations, Dr. *Carter* has succeeded in communicating "spirillum fever" to monkeys by inoculation with spirillar blood. Forty-four trials were made upon fifty-one of the common small Indian monkeys (Lemnopithacus Entellus and Macacus radiatus); defibrinated blood was injected subcutaneously. Of thirty-one inoculations made with spirillar blood, twenty-two were successful, and nine failed; all nine inoculations of non-spirillar blood and seven of desiccated blood (mostly infective when fresh) gave negative results; four injections made with saliva of fever patients and a healthy man produced serious, but not specific results. *Carter* found that intensity of infection increases by repetition, and, as in man, the blood at invasion-attacks is not so active as that of relapses, so the monkey's blood, at second remove from man, was found to produce a fatal result in the two cases in which it was used. *Motschutkoffsky's* experiments upon men led him to a somewhat different conclusion. *Carter's* investigations will be published more fully, but the main conclusions arrived at are stated as as follows: (1), that the spirillum or relapsing fever of man is directly transmissible to a quadrumanous animal; (2), that there occurs a non-febrile infection of the blood prior to "fever;" (3), that, though the blood spirillum was never seen in the monkey without fever ensuing sooner or later, yet the pyrexia is secondary in time, and is susceptible of highly varied manifestations. *Carter* has now succeeded in cultivating the spirochæte outside the body.

Dr. *Koch*, of Wollstein, at whose instigation we believe *Carter* made his experiments, has likewise succeeded in cultivating spirochætes, pursuing the same methods adopted by him with the bacillus anthracis of malignant pustule, and has also succeeded in inoculating monkeys with relapsing fever. One of these was killed during the active period of the disease, and the spirochætes were found in the various organs and in the skin. This result has never been reached with the human body, and should be reaffirmed.

Dr. *Lewis*, of Calcutta, who studied "spirillum fever" in India simultaneously with Dr. *Carter*, in his lately-published results, does not agree with the latter either in his observations or his conclusions. He regards the spirillum merely as a coincidence, and as no more essential to relapsing fever than is famine.

Prof. *F. Cohn*, of Breslau, however, finds serious defects in *Lewis'* methods of microscopical research, which he thinks quite invalidate the conclusions arrived at, and he is unwilling to regard *Lewis* as an authority. The observations which are published which indicate a positive connection between the parasite and the disease are certainly far more convincing than any negative results we have seen.

Holsti says relapsing fever was first recognized in Finland, in 1865, and was probably introduced from Russia. In 1866 it was accompanied by typhus. In 1867 it began to diminish in Russia, but this was a disastrous year for Finland, on account of a total failure of crops. With famine associated itself an outbreak of relapsing and typhus fevers, of such extent and severity that in 1868 about a third of the population was attacked by one or other of these maladies. In 1869–70 relapsing fever still existed in Helsingfors, but was milder and more rare, after the latter year disappearing altogether. At the beginning of August, 1876, it appeared in Helsingfors afresh at two quite distinct points simultaneously. It was impossible to trace its introduction from without. The epidemic developed rapidly, reached its culminating point in November and December, and diminished slowly, so that the latter half of 1877 presented only a few sporadic cases. There were about 600 cases during this epidemic, and of these *Holsti* saw 437. The disease confined itself almost exclusively to the poorest classes, occupying dirty crowded dwellings. One collection of habitations occupied by the most wretched of the urban population furnished 146 cases of the disease out of 560 inhabitants. The general rate of mortality was large, 7.8%. In 20% of the total there was but one febrile crisis, about the same proportion had two or three crises, occasionally there were four, five, and even six separate attacks; the first attack lasted as an average 6 days; the second, 3 to 6, and the third only 3 days. The average duration of the first intermission was 7.2 days, that of the second 8.4.

The blood was examined for spirilla in forty cases, and the organism was always found. They appeared generally one or two days after the commencement of the fever, and disappeared shortly before its termination. They could not be found during the intermission. This writer states that they were most abundant during the first access of fever. He could not establish any correlation between the intensity of the attack and the abundance of the spirilla in the blood; nor could they be traced in other secretions of the body, as the saliva, sweat, or urine. He reports as little success in cutting short the attacks with salicylate of soda as do other experimenters.

Dr. *Enke's* report of 136 cases which presented themselves in the Hospital at Magdeburg offers testimony of a positive character. He says spirilla were found in every case without exception, in some cases, indeed, only after persistent and repeated examinations. In a few instances where relapsing fever was suspected, it was only after as many as eight preparations of the blood were made that spirilla were found. *Enke's* conclusion is, that spirilla are to be regarded, not simply as an accompaniment, but as a cause of this disease.

Dr. *Riess* took advantage of a mild epidemic of relapsing fever, which developed itself in Berlin early in 1879 and dragged on through the year, to investigate with all possible care the connection between spirochæte and the changes in temperature. His results are based upon 77 cases of undoubted relapsing fever, and emphasize somewhat the difficult nature of this inquiry, and the caution necessary in drawing conclusions. *Riess* regards his cases as typical relapsing fever, but acknowledges that in a considerable number there was a certain irregularity and indistinctness about the relapses. This may explain his results where they vary from those of others. He observed a striking want of congruity between the appearance of spirilla and elevations of temperature.

The spirilla were seldom constant during the height of the relapse, one day spirilla were found and another day none. The length of time elapsing from the beginning of a relapse to the appearance of spirilla varied much; they appeared on an average from a half to one day later than the first rise in temperature; their behavior toward the close of the relapse was equally varied, occasionally not disappearing until some hours after the crisis. Not unfrequently no spirilla at all could be found during an entire pyretic period; out of 96 pyretic periods they were absent 36 times. Again in a few cases no spirilla could be found during any stage of the entire course of the disease.

THE PLAGUE.

BIBLIOGRAPHY.

J. Netten Radcliffe: On the Prospect of a Reappearance of Plague in this Country and on the Continent of Europe. Med. Times and Gazette, I., p. 83, 1876. —A. Hirsch: Was hat Europa in der nächsten Zeit von der orientalischen Pest zu fürchten? Deutsche Vierteljahrschrift f. öffentl. Gesundh., VIII., S. 377, 1876. —The Same: What has Europe to Fear in the Future from Oriental Plague? London Practitioner, XIX., p. 223, 1877.—Colvill: Plague in the Province of Bagdad, 1876–77. Rep. Med. Off. Local Gov. Bd., 1877, pp. 143–146, Lond., 1878. J. N. Radcliffe: Memorandum on the Progress of Levantine Plague in 1877 and part of 1878, together with an Account of an Outbreak of the Bubonic Plague of India (Máhámari) in 1876–77. Rep. Med. Off. Local Gov. Bd., 1877, pp. 139–157, London, 1878.—Castaldi: Plague in Resht, 1877. Rep. Med. Off. Local Gov. Bd., 1877, pp. 147–180, Lond., 1878.—Adler: Die Pest in Bagdad in den Jahren 1874–76. Allg. Wien. med. Ztg., XXIV., S. 32, 43, 55, etc., 1879.—Döppner: The Plague in Russia. (Transl. of the first official medical report of the disease in Astrakhan). Lond. Lancet, I., pp. 173–175, 1879.—The Pest in Russland. Wien. med. Wochenschr., XXIX., S. 65, 1879.—A. Hirsch: Ueber die Beulenpest. Berliner klin. Wochenschr., XVI., S. 85, 1879.—De Lesseps: Sur les mesures prises par l'Intendance Sanitaire de Marseille, dans la Crainte de l'Invasion par la Peste. Compt. rend. Acad. d. Sc., Paris, LXXXVIII., 324–327, 1879.—J. N. Radcliffe: A Short Account of the Progress of the Plague from its Reappearance in 1853. Med. Times and Gaz., Lond., I., p. 251. 1879.—Virchow: Ueber die Pest. Berl.

klin. Wochenschr., XVI., S. 117, 230, 1879.—Plague: Papers relating to the modern History and recent Progress of Levantine Plague. Prepared from time to time by direction of the President of the Local Government Board, with other papers, etc. (Presented to both Houses of Parliament by command of her Majesty, 1879). London, 1879.—HIRSCH: Mittheilungen über die Pest-Epidemie im J. 1878–9 im Gouvernement Astrachan. Berl. klin. Wchnschr., XVI., 445, 465, 1879.—Abstract of Lectures on the Plague Epidemic in the Year 1878–79 in Astrachan. Delivered before the Berlin Med. Soc. by PROF. HIRSCH, Member of late Germ. Plag. Com. Lond. Med. Times and Gaz., II., pp. 359, 416. 1879.—M. SOMMERBRODT: Zur Symptomatologie der "Pest von Wetljanka." Berlin. klin. Wchnschr., XVI., S. 477. 498, 1879.—THOLOZAN: Plague in the Caucasus; a Study in Sanitary Science, Lond. Practitioner, XXIII., 224, 1879.—THOLOZAN: Les trois Dernières Epidemies de Peste du Caucase, chronologie, géographie, prophylaxie, Par., 1879.—The British Commissioners' Report on the Plague. Brit. Med. Jour., II., 515, 1879.—ED. DICKSON: Résumé du Rapport sur la Peste de 1876–77 en Mésopotamie. Gaz. Méd. d'Orient. Constantinople, XXII., p. 49, 1879.—J. C. WISE: Plague. Hyg. and M. Rep. U. S. Navy, IV., 777, Wash., 1879.—PETRESCO: Mémoire sur l'Epidémie de Peste du Gouvernement d'Astrakhan. Bucharest, 1879.—Second Annual Report of the State Board of Health of the State of Connecticut. Hartford, 1880. —The Plague on the Volga: Reports of the German Medical Commission. London Lancet, I., p. 613, 1880.—Mittheilungen über die Pest-Epidemie im Winter 1878–79 im russischen Gouvernement Astrachan. Berlin, 1880.—The Plague in Russia. Brit. Med. Jour., I., p. 91, 1880.

The continued outbreak of epidemics of plague of greater or less magnitude at various points, some of them widely separated, since the one of 1871 in Persian Kurdistan, the last-mentioned in the American translation of *v. Ziemssen's* Cyclopædia, have impressed still more strongly upon the medical world the fact that the plague is still an active disease, especially in the Ottoman and Persian dominions; and the actual appearance of the disease in an epidemic form in south-eastern Russia has shown that the warning given by certain distinguished epidemiologists of a possible danger therefrom, under certain circumstances, to Europe itself was not uncalled for. In 1875, and again in 1876, Mr. *Netten Radcliffe*, in papers read before the "Society of Medical Officers of Health" in England, discussed the prospect of reappearance of plague in that country and in Europe. In 1876, Prof. *A. Hirsch* brought the same subject to the notice of the profession in Germany.

We give a brief summary of the outbreaks of plague succeeding that in Persian Kurdistan in 1871, for which we are indebted largely to these two writers. The plague appeared in Kurdistan in the winter of 1870–71, in a mountainous part of a district which is said to have been free from the disease for forty years; it continued throughout the greater part of 1871, and out of 7,000 people it was found to have affected at least 1,120, of whom 891 or 79.5% died. The next outbreak of plague occurred two years later in the winter of 1873–74, when the disease reappeared in Mesopotamia, on the lower Euphrates in the same district attacked in 1867, but on the opposite side of the river. This outbreak was more severe than the preceding one; the disease prevailed actively during the first six months of 1874, and it is estimated to have caused 4,000 deaths among a population of 80,000, appearing afresh in the winter of 1874–75, and lasting until the summer of that year. In all these countries, owing to the tendency to concealment to avoid isolation and quarantine, the number of cases and of deaths is more apt to be under- than overestimated. In March of 1874, plague broke out among the highlands of the Assyr district of Arabia, extending to within a short distance of Mecca. In April of this year it again declared itself among the nomadic tribes of the hilly district of Bengali in Tripoli. In 1876 we find plague again devastating the Euphrates valley, during which year it is estimated that not less than 20,000 persons were killed by it in this district. It ravaged Bagdad during this and the following year, carrying off more than 5,000 victims. Already in 1876 it had reached south-west Persia, later in this year appearing in north-western India, and about the same time in northern Persia, in the Province of Azerbijan and in Astrabad, from which it extended in 1871 to the Province of Ghilan on the Caspian Sea, attacking especially the principal town Resht, which has an active commerce with Astrachan, at the mouth of the Volga; it had been utterly desolated by plague in 1830, having then a population of about 40,000. Before the close of the year 1877 the plague is estimated to have killed about 4,000 people in this town. Cases of the disease are said to have occurred during this year at several places on the Caspian littoral of Caucasia, and it is also said that a number of cases were reported to the medical department of the Russian government as occurring in south-western and north-eastern Russia; but in regard to this the information is not very definite. Quarantine against importation of plague from Persia was not established on the Russian coast until April, 1877. From July to September, 1877, about 150 cases were observed in the town of Astrachan and the surrounding places of a disease which showed a resemblance to a mild form of plague, or was actually considered as such. According to *Hirsch*, the disease manifested itself chiefly by swelling of the lymphatic glands, the tumors being seated for the most part in the submaxillary, and also in the axillary and inguinal glands. A febrile state of variable duration, lasting even fourteen days, preceded the swellings in some cases. These swellings varied much in size; often they resolved, sometimes they suppurated; but with the exception of only one case, where the tumor became septic, and death followed by pyæmia, all these cases terminated favorably. Only a few patients were confined to bed. In no instance whatever were persons coming in contact with these patients infected, a fact which was verified especially in the hospitals; the garrison of the town also escaped entirely. These cases were seen by Dr. *Döppner*, the principal medical officer of the Cossack forces in the Province of Astrachan, and the description quoted corresponds with that given by him of the early cases in the subsequent epidemic at Wetljanka, with that given by Dr. *Cabiadis* of the cases of glandular swellings free from fever which preceded the severe outbreaks of plague at Bagdad and Hillah in 1876–77, and also with the symptoms and course of a few sporadic cases observed in several parts of Russia in 1879, but especially by Prof. *Botkin* in St. Petersburg. These cases in St. Petersburg gave rise to much discussion and difference of opinion, but Prof. *Botkin* stoutly maintained that they were the result of a mild form of the plague poison, and in this view was supported by others.

This mild type of plague—if such it was—which showed itself in the town of Astrachan in the summer of 1877, was followed in October of 1878 by the appearance of a similar malady at Wetljanka, a small stanitza or Cossack village, situated on the right bank of the river Volga, about one hundred and fifty miles from Astrachan, the inhabitants of which are for the most part devoted to fishing. This mild form rapidly developed into a disease of a malignant and fatal character, exhibiting all the recognized features of the oriental bubonic plague. From its comparative proximity to the termination of an important line of railway, this outbreak attracted the attention and excited the interest, not only of Europe and England, but even of this country, and we shall, therefore, give a somewhat more detailed account of it.

Whether the poison causing this outbreak had an autochthonous origin or was imported, and if imported, how, must remain undecided. The data on which to form an opinion are derived from the reports of Dr. *Döppner*, the surgeon-general of the district, and from those of the commissioners of the different European Governments appointed to serve as an International Commission for the investigation of the plague in Russia. It is not easy to reconcile the sometimes conflicting statements of these authorities; Prof. *Hirsch* and Dr. *Döppner*, for example, differing as to the distance of Wetljanka from Astrachan by fifty-six versts or about thirty-seven miles. Dr. *Döppner* labored under the disadvantage of occupying an official position and having to justify a previous course of inaction; the commissioners, under that of arriving on the scene independently and after the termination of the epidemic. The commissioners, moreover, had to carry on their inquiries by means of interpreters. Dr. *Colvill*, of the British Commission, was disposed to adopt the theory of an "independent origin" of the epidemic—and his previous experience of the disease in the Euphrates Valley lends weight to his opinion—autochthonous in the sense of indigenous, not in that of *de novo*, as explained by his fellow-commissioner Dr. *Payne*.

The most probable origin under the circumstances would seem to have been in importation, either from Astrachan or from some of the fortified towns, as Erzeroum, Kars, Bayazid, in eastern Asiatic Turkey which were besieged and sacked by the Cossack troops. The belief of the great majority of the Commissioners, including Prof. *Hirsch*, the head of the German Commission—a belief shared by the parish priest at Wetljanka, by the local authorities, and by Count *Melikoff*, sent as Governor-General of the Province—was that the poison was imported in packages of goods which reached Erzeroum or Kars from places in Persia or Mesopotamia, already infected, remained unpacked in the bazaars, and falling into the hands of Cossacks were sent by them as booty to Wetljanka.

Prof. *Hirsch* mentions a very clear case of infection with plague by means of clothing brought in a box from a house where people had died of the disease to another house at a distance, which fell under his own observation.

As far as its situation and sanitary conditions are concerned, Wetljanka seems in no respect to favor the prevalence of zymotic diseases any more than any of the other eighteen stanitzas of this district. The last time plague visited this village was in the year 1807–08; it was then supposed to have been brought by pilgrims from Mecca; it spread along the Volga as far as Sarotov; there were but few cases, and the mortality was small, not above one hundred for the whole district. The following account of the epidemic of 1878–79 is in the main that of Prof. *Hirsch.* It began in October, spread from Wetljanka as a centre, to six or eight villages on both sides of the Volga, and was practically at an end by the first of February.

The progress of its development in Wetljanka may be observed from the table of mortality which is accurate up to the middle of December, at which time the parish priest, who kept the records, succumbed to the disease:

	Deaths.
From Oct. 1 to Oct. 7	1
" " 8 " 14	0
" " 15 " 21	2
" " 22 " 28	3
" " 29 to Nov. 4	0
" Nov. 5 " 11	1
" " 12 " 18	7
" " 19 " 25	8
From Nov. 26 to Dec. 2	7
" Dec. 3 " 9	56
" " 9 " 16	169
" " 17 " 23	54
" " 23 " 30	33
" " 31 to Jan. 6	19
" Jan. 7 " 14	12

On the whole there were 373 deaths at Wetljanka during the whole period. By subtracting the average number of deaths that occur (according to an average of several years) from October till January in that place, viz., fourteen, there remained 359 deaths from plague; in other words, in a population of about 1,750 individuals, 20 per cent of the whole perished with the disease. It is very difficult to arrive at the number of cases that recovered. According to very barely trustworthy statements, this was said to have been 81. If those are added to the 359 deaths, we have a morbidity of 440—that is, 25.3 per cent cases of illness amongst the inhabitants, with 82 per cent deaths of the total number attacked.

After Wetljanka, Prischib was first attacked with the disease. Here the number of deaths was 16, which occurred in five houses. Afterwards there was a small outbreak of the disease in Staritzkcje, where 7 individuals succumbed in one house. On the left bank two places had only a few cases, namely, Michailowka, where in one house 4 cases occurred with 3 deaths, and Udatschucie, where in one house 2 individuals died of plague. Selitroucie suffered the most severely; here in four houses 32 individuals succumbed to the disease. It was said that in the steppe two plague-cadavers had been found, and that opposite to Wetljanka, in an island in the Volga, several mortal cases of plague had occurred amongst the inhabitants of Wetljanka who had fled there. It is impossible to make out whether those statements were well founded or not, but it might be not far from the truth if the total number of deaths from plague in the district were estimated at 450.

About Dec. 18th a cordon was drawn around Wetljanka and toward the beginning of January a general cordon of five thousand soldiers around the whole infected district, with quarantine stations at the north-western and south-eastern extremities on both sides of the river. The quarantine arrangements are reported to have been very imperfect, and the speedy termination of the epidemic is not attributed especially to these dispositions.

SYMPTOMATOLOGY.

The various descriptions of plague which have been given by those having personal experience of it during its greater frequency within the last five years agree in most particulars with the account given by *Liebermeister* in the first edition of *v. Ziemssen's* Cyclopædia. It is greatly to be regretted that the Russian Government did not promptly avail itself of the favorable opportunities afforded by the epidemic at Resht and in the provinces of Astrachan to have the symptomatology and pathology of the disease carefully investigated by competent persons, whose services it could easily have commanded. No post-mortems were made during the Wetljanka epidemic. The following analytical statement of 1826 cases of plague noted by Dr. *Giovanni Cabiadis*, at Hillah, during the epidemic of 1876, is taken from an account of the characters of epidemic plague in Mesopotamia in 1876–77, prepared by Dr. *Dickson*, Physician to the British Embassy at Constantinople, from Dr. *Cabiadis'* notes. Dr. *Cabiadis* occupied an official position under the Persian Government and had every opportunity for observation.

As a rule, an attack of plague lasts from a few hours to four weeks, and Dr. *Cabiadis* considers that one-third of the attacks end fatally. From the accompanying table, however, 1,826 cases of plague seen and registered by him at Hillah, in 1876, the proportion of deaths to that of the attacks is 52.6 per cent. But a great many cases of plague, he observes, are never made known, and would thus diminish very considerably this percentage of its mortality; whilst the deaths, on the other hand, must all be reported in order to obtain the permit of interment.

ANALYTICAL STATEMENT OF 1,826 CASES OF PLAGUE NOTED BY DR. CABIADIS, AT HILLAH, DURING THE EPIDEMIC OF 1876.

From 2 months to 9 years,	277
" 10 years " 19 "	617
" 20 " " 29 "	432
" 30 " " 39 "	292
" 40 " " 49 "	123
" 50 " " 59 "	52
" 60 " " 69 "	18
" 70 " " 79 "	11
" 80 " " 89 "	3
An old man of 113	1
Total,	1,826

Sex: Male, 889; Female, 937; total, 1,826.
Result: Recovered, 865; died, 961; total, 1,826.

Manifestations:—

Glandular swellings—in the groin,		710
" " axilla,		466
" " neck,		98
" " several places,		122
		1,396
Carbuncles,		36
Dependent on the nervous centres	Coma,	28
	Convulsive shake,	9
" " circulating system	Petechiæ,	120
	Epistaxis,	2
	Hæmoptysis,	6
	Hæmatemesis,	27
	Sanguineous diarrhœa,	14
	Menorrhagia,	2
" " assimilative organs	Bilious vomiting,	32
	Bilious diarrhœa,	16
	Jaundice,	2

Dr. *Cabiadis* does not attempt to deny the contagious properties of plague, but the experience acquired by him during the outbreaks at Hillah (the ancient Babylon) and Bagdad has convinced him that no great risk is incurred in touching a person affected with the plague, provided exposure for any lengthened period to the atmosphere of the apartment occupied by him is avoided. He says that those persons who lived in the same house with a plague-smitten patient, but who avoided touching him or his clothes, through fear of catching the infection, generally did get the plague; whilst those who lived in houses exempt from the malady, but who visited plague-patients and handled them freely, without remaining long in the same room with them, hardly ever caught the disease. *Colvill's* description of the symptoms of plague, as observed by him on the lower Euphrates in 1875, agrees with that of Dr. *Cabiadis* in the main. Both of these observers mention the vomiting and spitting of blood, and hemorrhages from various surfaces as not unusual. This was doubtless the same disease which invaded Wetljanka, and in this respect these accounts agree with those of *Döppner*, an eye-witness, and of *Colvill* of the epidemic at that town in 1878–79. *Sommerbrodt*, *Hirsch's* colleague on the German commission, says: It cannot be strictly denied that at a certain period of the epidemic pulmonary symptoms were prominent. Prof. *Hirsch's* account of the symptoms in the Wetljanka epidemic differs somewhat from those mentioned, and we quote what he says:

> I do not intend to discuss at present the question whether, as some maintain, the epidemic plague known by the name of the "black death" and the so-called "Indian plague" are identical with the Levantine bubonic plague; or whether they differ from each other, as I have assumed and still assume, by definite symptoms which reveal an affection of the lungs. Perhaps at another time I may re-

turn to that question; to-day I have only to say that the lung affection named had not the character of pneumonia, but of pneumorrhagia: and that in the course of the disease in the Astrachan epidemic no such complication either with pneumonia or with bleeding from the lungs could be observed. In a few cases only slight bronchitis seems to have been observed as a complication. Moreover, I will not deny that cases of pneumonia occurred at the time of the epidemic; only I do not believe that they had anything in common with the plague. As in all former plague epidemics, there are three forms to be distinguished, according to the degree of development of the disease. The first form is slight, partly without fever, a sort of ambulatory plague; where without any other grave symptoms, a bubo was formed in the axillary, inguinal, or submaxillary glands, which soon suppurated or resolved, and where the whole process was terminated in that way. Cases of this form seem to have been more frequent, especially in the beginning and towards the end of the epidemic. Of those we saw ourselves a few, as I have just said. But, secondly, there was a series of cases of a graver character, where buboes were formed after an illness of several days, and where the whole organism was deeply affected. Most of the buboes in those cases became purulent, and recovery was only exceptional. A third series of cases was observed in the form of the so-called *pestis siderans*, where no localization of the morbid process in the lymphatic system was formed at all, or the buboes were so small that they escaped the attention of the surgeons and the friends of the patients. Those cases proved fatal without exception, in two or three days.

Under the protection of Prof. *Virchow*, it may not be rash to express a regret that *Hirsch* should have introduced an apparently unnecessary element of confusion into the study of the disease. Plague is probably plague just as typhoid fever is typhoid fever all the world over. It is very evident from the accounts of the various epidemic outbreaks that soil and circumstances modify the plague poison and its manifestations as they do those of typhoid fever, so that some symptoms, and notably buboes and hemorrhages, vary in frequency and severity in different epidemics and at different periods of the same.

A frequent affection of the lungs hardly requires a different name, as "Indian plague," any more than we should now speak of "black death," because the hemorrhages in the skin were unusually large or confluent. The erection of the Pali plague, and the endemic disorder observed in a small district in the Himalayas near Nepaul, into a distinct disease under the name of Indian plague savors somewhat of the "*Schreibtisch.*"

According to *Radcliffe*, who follows *Planck*, Máhámari (the "great plague," a term also applied to cholera) and locally designated gola or phutkia (bubo)—the several terms being generally used in the sense of pestilence—seems to have been first noted in Cutch, in 1815, and during the six years 1815–1820 the disease prevailed in parts of Cutch, Kattywar, and Sindh. In 1823 the malady is reported to have appeared among the Himalayas, in the district of Garhwál, a district lying to the north of and contiguous to Kumaun. In 1834–5 it was prevalent in parts of Garhwál. In 1836 the disease broke out at Pali, in Marwar, Rajputana (whence the name Pali plague), and it subsequently prevailed in several parts of the Meywar and Marwar districts. From 1847 to 1853 Máhámari seems to have been present in a more or less active form in, or at least rarely absent from Kumaun, and in the last-named year the malady was also observed in Rhilcund, the district lying immediately to the south of Kumaun. In 1859–60 the disease was again active in both Kumaun and Garhwál; also in 1870. From 1870 to the outbreak of 1876–7, in Kumaun, there does not appear to have been any record of an appearance of the disease. In 1876–77 forty-one of the hill villages in the mountainous district of Kumaun, on the southern slopes of the Himalayas, suffered from this malady; in forty of these villages two hundred and ninety-one persons were attacked, of whom two hundred and seventy-seven died. Mr. *C. Planck*, the Sanitary Commissioner for the North-Western provinces and Oudh, inspected the infected district at the time of the prevalence of this outbreak, and has given a detailed account of the results of his inspection. He regards this disease as the same as the Pali plague at Rajputana and contiguous provinces to the westwards—so likewise does *Hirsch*—and, moreover, identifies it with oriental or Levantine bubonic plague. *Planck* says: The symptoms and character of this disease coincide very exactly with the description of plague, with the exception that the appearance of petechiæ (purple spots) on the skin has not been noticed in the Kumaun form of the disease. No appearance of that nature could be seen on the skin of the sick persons examined, and no evidence of such appearance in any case could be obtained. He attributes this absence of visible petechiæ to the fact that the people examined were all dark-skinned. For a more detailed description the reader is referred to p. 48 of "Papers Relating to the Modern History and Recent Progress of Levantine Plague, London, 1879."

The most palpable and evident of all the causes which predispose an individual to an attack of plague during an epidemic Dr. *Cabiadis* considers to be poverty with its accompaniments. In his experience he found that the poor were seldom spared; the wealthy hardly ever attacked; in this respect presenting a contrast to cholera.

TREATMENT.

Recent epidemics have added nothing whatever to our knowledge upon this point; the treatment is purely expectant.

Prophylaxis.—According to *Cabiadis*, the most effectual means for the protection of a community against the propagation of plague are the isolation of the sick, the destruction by fire of their clothes, and the whitewashing with lime and free ventilating of the domiciles in which cases of plague occur. The plague reappeared in only one out of three hundred and fifty houses which had been *whitewashed* after an outbreak of plague in them, whilst its reappearance in houses which merely had been abandoned for a time, after an attack of that disease, but which had not been whitewashed, *was* of frequent occurrence. The other disinfectants tried, such as sulphur fumigations, lighting fires, and throwing a solution of sulphate of iron into the drains, gave no decided results, except when associated with the whitewashing and airing of the infected houses.

v. Pettenkofer recommended sulphuric acid as a disinfectant, as he found it the most reliable means of destroying the bacteria of charbon. *Virchow* thinks dry heat would prove more reliable in disinfecting effects.

All recent writers agree with *Aubert* (De la Peste ou Typhus d'Orient, Paris, 1840) that civilization alone has destroyed plague in Europe, and it alone will annihilate the disease in the East.

QUARANTINE AND CORDONS SANITAIRES.—The late epidemic in the province of Astrachan is but little instructive in regard to the value of these measures, for the epidemic was already at an end when these steps were taken. *Hirsch* concludes his remarks upon the Astrachan outbreak as follows:

> The principal task of a public sanitary administration must lie, according to my conviction, in a strict supervision of the places which are menaced by the plague, and in a well arranged isolation of the first centres of the disease. If we should have learnt nothing more from that small plague-epidemic than to estimate sufficiently the value of such isolation of infected localities for avoiding further propagation, the victims have not fallen in vain.

The value of quarantine must vary with the conditions under which it is applied. For countries like England and the United States, quarantine against plague would be unnecessary if not impracticable. There are other countries where quarantine might be desirable if a strict one were possible, which the exigencies and temptations of modern commerce render very doubtful.

The rag-trade would be the most probable means by which plague might be introduced into the United States when prevalent in the Levant. New Haven is said to be the only port in this country that receives whole cargoes of rags, and these come for the most part from Egypt. After the outbreak at Wetljanka, measures were taken by the National Board of Health for the disinfection of cargoes from infected ports. The Connecticut State Board of Health, in its second annual report, states that reports from the paper manufactories in the State, where the bales of rags are unpacked, fail to show the communication of any other disease than small-pox by rags, and that by domestic rags in a few instances.

A proper distinction is to be made between medical inspection, disinfection, and cleansing as practised to-day and in this country, and the exercise of measures hitherto implied by the term quarantine, in regard to which we may quote the language of Mr. *John Simon* in his eighth annual report: A quarantine, which is ineffective, is a mere irrational derangement of commerce, and a quarantine, of a kind which insures success, is more easily imagined than realized. Only in proportion as a community lives apart from the great highways and emporia of commerce, or is ready and able to treat its commerce as a subordinate political interest, only in such proportion can quarantine be made effectual for protecting it. In proportion as these circumstances are reversed, it becomes impossible to reduce to practice the paper-plausibilities of quarantine. The conditions which have to be fulfilled are conditions of national seclusion, and the fulfilment of such conditions by England (or the United States) would involve fundamental changes in the most established habits of the country.

YELLOW FEVER.

BY

GEORGE M. STERNBERG, M.D.,

SURGEON U. S. ARMY.

YELLOW FEVER.

BIBLIOGRAPHY.

Dagnino, M.: De la fiebre amarilla, ensayo pratico sobre esta enfermedad observada en Maricaba, en la Republica de Venezuela. Genoa, 1873.—Faget, J. C.; Type and Specific Character of True Yellow Fever. New Orleans, 1873.—Hargis, R. B. S.: Yellow Fever Epidemic at Pensacola in 1873. N. O. M. and S. J., 1873, p. 781.—Jones, J.: Temperature in Yellow Fever. Boston M. and S. J., 1873, LXXXIX., p. 205. Contributions to the Natural History of Yellow Fever. N. O. M. and S. J., 1873, N. S., I., p. 465. Yellow Fever Epidemic of 1878 in New Orleans. N. O. M. and S. J., 1879, N. S., VI., p. 599.—Russell, S. C.: Yellow Fever, Rep't B'd of Health, La., 1873.—Le Monnier, T. R.: Yellow Fever Epidemic of Memphis, Tenn., in 1873. N. O. M. and S. J., 1873, I., p. 449, 536, 656, —Saunders, D. D.: Observations on the Yellow Fever Epidemic of 1873, at Memphis, Tenn. N. O. M. and S. J., 1873, I., p. 791.—Bérenger-Féraud: De la fiévre bilieuse mélanurique des pays chauds comparée avec la fiévre jaune, étude clinique faite au Sénégal. Paris, 1874. De la fiévre jaune á la Martinique. Paris, 1878.—Cochran, Jerome: The Yellow Fever Epidemic of 1873. Tr. M. Ass., Alabama, 1874.—Report of Com. of the Shreveport Med. Soc. on the Yellow Fever Epidemic of 1873. Shreveport, 1874.—Herron, J. S.: Yellow Fever at Pensacola in 1874; Yellow Fever at Barrancas, Fla., in 1875. A. Report Sup. S. G. Marine Hospital Service, 1874 and 1875.—Reilly, F. W.: The Yellow Fever Epidemic of 1873. R. Sup. S. G. Marine Hospital Service, 1874.—Tryon, J. R.: Epidemic of Yellow Fever at the Navy-Yard, Pensacola, Fla., in 1874. San. and Med. Reports, Navy Department for 1873-4, p. 451.—Wucherer, O.: Einige Bemerkungen über das Gelbfieber und seine Verbreitungsweise. Deutsches Arch. f. klin. Med. Leipzig, 1874, XII., p. 391.—Bayley, R. A.: Report on Yellow Fever Epidemic at East Pascagoula, Miss., in 1875; A. Rep. La. Board of Health, New Orleans, 1876.—Herrick, S. S.: Review of Yellow Fever in New Orleans, 1869–74, N. O. M. and S. J., 1875, II., p. 645.—Murray, R. D.: Yellow Fever at Key West, Fla., in 1875; A. Rep. Sup. S. G. Mar. Hosp. Service, 1875.—White, C. B.: Disinfection in Yellow Fever as Practised in New Orleans in the Years 1870 to 1876 inclusive; Am. Pub. Health Assoc. Repts., 1875–6, p. 154.—Sternberg, G. M.: On the Nature and Duration of Yellow Fever, Am. J. M. Sc., 1875, LXX., p. 99; Yellow Fever in Pensacola, Fla., in 1873–4–5; Am. Pub. Health Asso. Repts., New York, 1876, II., p. 468; A Study of the Natural History of Yellow Fever, N. O. M. and S. J., March, 1877, p. 638.—Walkly, N.: On the Nature and Treatment of Yellow Fever, N. O. M. and S. J., 1876, p. 15.—Reyna y Puyon, M. de: El Tifus icterodes ó Fiebre Amarilla, Madrid, 1876.—Plata, M. de la: Eysayo de una Descripcion Comparation entre la Fiebre Amarilla y la Bilosa Grave de los paises cálidos, Crón. Méd. Quir. de la Habana, 1876, II., pp. 215, 262, 334.—Brendel, C.: Beobachtungen über Gelbfieber in Montevideo, Deutsche Vrtljschr. f. Off. Gsundhtspflg. Braunschweig, 1877, IX., p. 224.—Crevaux, J.: Notes sur l'Histologie Pathologique de la Fiévre Jaune, Arch. de Méd. Naval, Paris, 1877, XXVIII., p. 223.—Rey, H.: Notes sur la Fiévre Jaune au Brésil d'aprés les Publications Récentes des Médecins Brésiliens, Arch. Méd. Nav., Paris, 1877, XXVIII., pp. 277, 372, 438.—WOODHULL, A. A.: On the Causes of the Yellow Fever Epidemic at Savannah in 1876, Am. J. M. Sc., Phila., 1877, LXXIV., p. 17.—CUNISSET: Etude Chemique de la Fiévre Jaune, Arch. de Méd. Naval, Paris, 1878, XXIX., p. 188.—ESPINA P.: Fiebre Amarilla, Bol. Med. Nav., San Fernando, 1878, I., pp. 165, 201; 1879, II., pp. 45, 64.—HORSEY, C. W.: Yellow Fever at Fernandina, Fla., in 1877, Proc. Fla. Med. Assoc., 1878, p. 51.—MARVIN, J. B.: A History of the Diagnosis, Pathology, and Treatment of Yellow Fever, Louisville, 1878. —MINOR, T. C.: Report on Yellow Fever in Ohio in 1878, Cininnati, 1878. —SIMONS, M.: A Note on the Epidemic of Yellow Fever at Port Royal in 1877. Trans. South Carolina Med. Association, 1878.—FISHER, L. C.: Yellow Fever at Rio de Janeiro. Nat. B'd Health Bull., Washington, 1879, I., p. 115.—GUICHET, A.: La fiévre jaune à Madrid en 1878, Rec. de mém. militaires, Paris, 1879, XXV., p. 337.—GAMGEE, J.: Yellow Fever a Nautical Disease. New York, 1879.—HAVANA COMMISSION: Preliminary Report to Nat. B'd of Health. Supplement to Nat. B'd Health Bulletin. Washington, 1879.—HEINEMANN, C.: Beiträge zur Kenntniss des gelben Fiebers an der Ostküste Mexiko's. Arch. f. path. Anat., etc., Berlin, 1879, LXXVIII., p. 139.—HORTON, J. A. B.: In his Diseases of Tropical Climates. London, 1879, p. 120.—HOLT, J.: Appearance of Yellow Fever in New Orleans during Summer of 1879. N. O. M. and S. Jour.—KEATING, J. M.: The Yellow Fever Epidemic of 1878 in Memphis, Tenn. Memphis, 1879.—NASHVILLE IN THE EPIDEMIC OF 1878: Rep. of Board of Health, City of Nashville, 1879.—PORTER, W. H.: The Pathology of Yellow Fever. Med. Rec. New York, 1879, XVI., p. 519.—SCHMIDT, H. D.: Pathological Lesions in the Nervous System in Yellow Fever. Med. Rec., N. Y., 1879, XVI., p. 44.—SATTERTHWAITE: Yellow Fever. Med. Rec., N. Y., 1879, XVI., p. 401.—VANDEMAN, J. H.: Yellow Fever in Chattanooga in 1878. Nashville J. M. and S., 1879, XXIV., p. 47.—WOOLVERTON, T.: An Account of Yeliow Fever on board the Plymouth. Hyd. and M. Rep., U. S. Navy, 1879, IV., p. 689.—WARING, J. J.: The Epidemic at Savannah in 1876. Savannah, Ga., 1879.—THORNTON, G. B.: The Memphis Yellow Fever of 1879. Boston Med. and Surg. Journal, 1879, CI., p. 787. Also Transactions of the Med. Soc. State of Tenn., 1879.—BOARD OF EXPERTS: Conclusions of Board of Experts authorized by Congress to investigate the Yellow Fever Epidemic of 1878. Washington, 1879.—CHOPPIN, SAMUEL: History of the Importation of Yellow Fever into the United States, from 1693 to 1878. Rep. Am. Pub. Health Ass'n, Vol. IV., p. 190.—MCCLELLAN, ELY: A Study of the Yellow Fever Epidemic of 1876. Rep. Am. Pub. Health Ass'n, Vol. IV., p. 249.—MORRIS, JOHN: History of the Epidemic in Baltimore in 1876. Rep. Am. Pub. Health Ass., Vol. IV., p. 243.—WATKINS, W. H.: Yellow Fever in southwestern Louisiana. Rep. B. of H., La., 1879, New Orleans, 1880.—BEMISS, S. M.: Clinical Study of Yellow Fever. Am. J. M. Sc. April, 1880.—CAMERON, COL. J. F.: Camps and Depopulation of Memphis in the Epidemics 1878-79. Proceedings of Am. Pub. Health Ass. Sanitarian, Vol. VIII., p. 97.

NOTE.—The recent literature of yellow fever is so extensive and the space alloted to this article so restricted, that the author finds it necessary to omit from the bibliography a large number of interesting papers which have been consulted in the library of the Surgeon-General's office, but which, owing to the wealth of material, he has not been able to use in preparing this article.

HISTORY AND ETIOLOGY.

The volume of *v. Ziemssen's* Encyclopedia in which appears the brief, but excellent article on yellow fever by *Haenisch*, was published in 1874; but, as no references are made, in the bibliography given, bearing a later date than 1871, the author of the present article has decided to include he epidemic of 1873 in the historical sketch of recent epidemics, which e proposes to give in this place.

1873.—Yellow fever was brought to New Orleans in 1873 by the Spanish bark Valparaiso, which sailed from Havana June 15th, in ballast; arrived at the N. O. quarantine station June 24th; was detained two days, and came to the city June 26th. The first case was that of the mate of this vessel who was taken sick on board, July 4th, while she was lying at the wharf. The third and fourth cases were on the river steamers Belle Lee and Albert Pike, which were lying at the wharf next above the Valparaiso. But for the sickness and death of the mate of the Valparaiso, it is doubtful whether importation would have been traced to this vessel, and the origin of this epidemic, like that of many others in the United States, might have remained obscure, or have been ascribed to local causes. The fact that no other cases occurred on the Valparaiso is explained by the statement of the captain that his crew consisted of *acclimated Spaniards.* Total mortality in N. O., 225 (*Russell*). From New Orleans yellow fever was carried to Memphis by the river steamer Bee. Two men, sick with yellow fever, were landed from this boat, Aug. 12th; both died, one in a house in "Happy Hollow," the other at the station-house in Adams Street. *The disease only spread from the former* (*Le Monnier*). Estimated mortality, 2,000. River steamers from New Orleans also introduced the disease into the town of Shreveport, La., where the disease became epidemic about Sept. 1st. First case, Aug. 12th; mortality, 759 (Rep. of Com. of Shreveport Med. Soc.). On the 3d of September, a refugee from Shreveport arrived at the town of Calvert, Texas. He was taken sick on the 5th, and died on the 10th. The bedding of this patient was thrown on the roof of a little house at the foot of Main Street, and left for three weeks in the sun. An epidemic resulted; mortality 125 (*Coleman*). Upon the outbreak at Shreveport hundreds of citizens came pouring into the town of Marshall, Texas. Many were taken sick soon after their arrival; 36 deaths occurred in this place (*Pope*). The disease was also introduced to Cairo, Ill., and Louisville, Ky.; seventeen deaths occurred at the former, and four at the latter place; at Greenwood, La., four deaths occurred, and at various minor places, 27 (*Reilly*).

The epidemics at Pensacola, Fla., and at Montgomery, Ala., are traced to a different importation. The ship Golden Dream arrived in Pensacola harbor, June 10th; she had lost three of her crew from yellow fever in Havana, and eight *en route;* was detained at the quarantine station until July 3d, then admitted to pratique and anchored 500 yards from the central wharf, Pensacola. On the 2d of August, one of the new crew of this vessel, shipped 8 days previously, was taken sick; he died on the 5th of August, with black vomit. On the 6th, three cases occurred in the city, all in the same house, about two squares from the water. The tenant of this house had visited the Golden Dream, remaining on board for several hours (*Hargis*). An epidemic followed with a mortality of 62. The disease was introduced to Montgomery by two refugees from Pensacola; one located in ward one, and one in ward five. From the residences of these persons the disease radiated in every direction; mortality, 108 (*Michel*). The disease was introduced into Mobile, Ala., according to *Cochran*, by a refugee from Shreveport; according to *Gilmore*, from Pensacola; 18 deaths occurred. During the summer of 1873, 64 cases with 18 deaths occurred at New York; of these, 62 cases were removed from vessels at the quarantine station, and two reached the city by land. At Fort Jefferson, Fla., there were 37 cases, with 14 deaths; and at Fort Barrancas, Fla., 12 cases, and 3 deaths. An epidemic at Montevideo this summer caused 400 deaths (*Brendel*).

1874.—Yellow fever was again introduced into Pensacola harbor by the Spanish bark *Doce de June*, which arrived at the quarantine station May 29th, having one case of yellow fever on board, and having lost one *en route* from Havana. From this vessel the disease was communicated to others at the quarantine station, and one of these vessels, the Elmira Coombs, being admitted to pratique after five days' detention at quarantine, brought it to the city. Mortality, 118 (*Herron*). A severe epidemic also occurred at the Pensacola navy-yard, ascribed by Surgeon *Tryon* of the navy to importation from the quarantine station. Total mortality on naval reservation 55. The disease also prevailed at Pascagoula, Miss., and a few cases (20) occurred in New Orleans (*White*).

1875.—On the 27th of June the bark Von Moltke, from Havana, came to anchor opposite Fort Barrancas, Fla., and the following morning she proceeded to the Pensacola quarantine station, where she was detained, owing to the sickness of five of her crew with yellow fever. *Twenty-one days after*, an explosion of yellow fever occurred at Barrancas, 37 cases occurring in different parts of the garrison within five days from the commencement of the first case (July 18th). Total number of cases, 74, total mortality 29 (*Sternberg*). At Key West, Fla., 41 deaths are reported; disease believed by reporter to have been of local origin (*Murray*). At East Pascagoula, Miss., the brig St. Michael, from Havana, arrived June 28th; cases occurred on this brig July 10th; an epidemic was developed with a total mortality of 56. The disease also prevailed in the neighboring towns of Scranton and Moss Point (*Bayley*). In New Orleans, 95 cases occurred between Aug. 8th and Nov. 25th. The first cases are traced to the schooner Orloff, of Moss Point, Pascagoula River; and the limited number of cases is ascribed, with a strong appearance of probability, to the energetic measures of disinfection resorted to (*White*).

1876.—This year, after a complete immunity from the disease for eighteen years, a severe epidemic occurred at Savannah, Ga., causing a mortality of 896.

The exhaustive study of this epidemic by Surg. *Woodhull*, U. S. A., seems to prove that the *first cases* in Savannah this year were of local origin, that is, not traceable *directly* to other cases, or to exposure on infected ships. But for those who believe that the yellow fever poison may be introduced independently of persons, and is capable of self-multiplication external to the human body, when local conditions are favorable, the following facts given by *Woodhull* are quite sufficient to establish a strong probability of importation of the poison.

> "The condition of Savannah in the summer of 1876 may be epitomized as follows: On the west, north, and south-east, and, in a less degree, on the east, there were badly drained low lands that had been flooded by unusually heavy rains in June; on three sides, but especially on the west, large amounts of garbage were deposited near the city. The sewers gave off foul gases, sufficiently to attract general attention; the supply of the city water-works was drawn from contaminated sources. The soil of the city was probably saturated with the products of animal decomposition, and the wells from which much of the drinking water was drawn penetrated this soil. To the east, at an average distance of 700 yards, was the open and practically stagnant sewer, known as the Bilbo canal, and 400 yards further east was a rice plantation in full cultivation; 1,200 yards down the river were two vessels from Cuba with a certain amount of Cuban ballast; two other vessels, and another quantity of ballast, also from Cuba, were at the wharves on the north-west. About forty-five Spanish seamen and their bedding, last from Cuba, were present in the city. The average temperature was 82° F.; and the average relative humidity was not quite 71°."

At Brunswick, Ga., an epidemic was developed about the same time as in Savannah, and apparently from an independent importation. Several smaller towns in Georgia also suffered to a slight extent (*M'Clellan*). In New Orleans, the disease again prevailed to a limited extent, and it is believed by the reporter that its spread was greatly restricted by the liberal use of disinfectants. First case August 11th; total cases 74; deaths 35. Importation not traced (*Holt*). At Baltimore, Md., a local epidemic occurred, which is believed by the reporter to have been of local origin. The infected locality was a small peninsula bounded on one side by Lancaster St., and on the other three sides by water. "On this peninsula between two and three hundred persons were huddled together in small tenements, all the surroundings being of the most noisome character." First case Sept. 14th; number of cases 44; deaths 40 (*Morris*).

It should be noted that the admission of the local origin of the first cases in an epidemic is a very different matter from an admission of the *de novo* origin of the poison. This may have been introduced but a short time before the occurrence of early cases, or perhaps many years before, as at Havana and other places where the disease is now endemic. In a great commercial city like New Orleans or Baltimore, the failure to prove importation can hardly be accepted as proof of non-importation. It must nevertheless be admitted that there are strong reasons for believing that sporadic cases and minor epidemics have several times occurred in the former city independently of any fresh importation of the poison during the years of their occurrence. But, in the opinion of the writer, no satisfactory proof is to be found of the *de novo* origin of yellow fever in the history of this disease, as recorded, from the earliest times to the present day.

1877.—An epidemic occurred in Fernandina, Fla., causing a mortality of 75 in 478 cases among the white population, and 20 *deaths in* 934 *cases among the colored population.* Several vessels arrived from infected ports prior to the development of the epidemic and the disease is believed to have been imported (*Horsey*). An epidemic also occurred at Jacksonville, Fla., which the reporter believes to have been of local origin; yet he says: "Our proximity to Fernandina (in which city yellow fever was declared epidemic in the latter part of August), and the numerous channels of intercommunication by land and water, rendered it very difficult, if not absolutely impossible, to establish any system of quarantine entirely free from weak points." The first case occurred Oct. 27th; mortality 95 (*Daniel*). At Port Royal, S. C., 183 cases with 25 deaths are reported and attributed to importation by *Simons*. In New Orleans but a single case, and that imported from Havana, occurred during the year.

This complete immunity is ascribed by the president of the Board of Health to a rigorous winter, an efficient quarantine, and thorough disinfection of the vicinity where the imported case was discovered and died (*Choppin*).

1878.—The epidemic of this year will long be remembered as the *great epidemic* of 1878. The number of places invaded was 132; total number of cases reported 74,265; total number of deaths reported 15,934.

Memphis stands first in the mortality record, estimated at 5,000; New Orleans, second, 4,600; Vicksburg, third, 872, etc. (Appendix to Report of Board of Experts appointed by Congress). The origin of this extensive epidemic is traced by Dr. *Choppin*, Pres. of the La. Board of Health, to the steamer Emily B. Souder, which arrived from Havana May 23d, and was moored at the foot of Calliope St. "The first cases of yellow fever at New Orleans in 1878 were, undoubtedly, two of the officers of the above steamship, namely, Clarke, the purser, and Elliott, one of the engineers."

Infected centres were developed in the immediate vicinity of the houses in which Clarke and Elliott were sick, and cases of local origin occurred about the middle of July. The fact that five or six weeks elapsed after the arrival of the Souder and the death of Clarke and Elliott, before these local centres of infection were developed, presents no difficulties to Dr. *Choppin*, who is thoroughly convinced of the truth of the "germ theory." In accordance with this view, the germs being introduced early in the season when local conditions, especially as to temperature, were not very favorable to their increase, either remained dormant, retaining their vitality, or multiplied so slowly that the poison was not present in sufficient quantity to produce its characteristic effects until the first or second week in July. While yellow fever thus effected a lodgment in New Orleans and extended along the main routes of travel as far as St. Louis, Mo. (*Carondolet*), Cairo, Ill., and Gallipolis, Ohio, where limited outbreaks occurred; the cities of Pensacola and Galveston on the Gulf escaped entirely, protected, without doubt, by their rigorous quarantine regulations. A detailed account of this great epidemic would far exceed the limits of the present article, and as there can be no reasonable doubt that New Orleans was the centre from which the disease extended itself, it is here treated as a single epidemic. A few physicians, however, dispute this extension from a single centre, and claim that the disease had its origin in situ, from a combination of atmospheric and telluric causes, existing at the points where it prevailed. Thus, on the northern limit of the extension of the disease at Carondolet, a suburb of St. Louis, Dr. *G. H. Ford*, in an elaborate discussion of meteorological and local conditions, comes to the conclusion that yellow fever was of local origin at this place; and that the fact that the ill-fated steamer John Porter lay at the foot of Market St., one or two squares from the point where a cluster of cases occurred 43 days later, can have no relation to these cases, "as an incubation period of 43 days can scarcely be admitted." The latter proposition will be readily agreed to, but the deduction may be questioned. The evidence of local origin in this case is of the same nature as that depended upon for the support of this view elsewhere, *e. g.*, Fort Barrancas, Fla., in 1875; Savannah, Ga., in 1876; Jacksonville, Fla., in 1877; New Orleans, La., in 1878; and at Madrid, Spain, in the same year. As the epidemic last referred to is a very instructive one, the main facts will be given here. Madrid is a well-built and healthy city, in the interior of Spain, 675 *metres above the sea level.* It is well drained and has an abundance of pure water. A circumscribed epidemic of yellow fever occurred in this city during the summer of 1878. In all there were 50 cases, with 35 deaths. The first case occurred Sept. 1st. At this time yellow fever was not prevailing at any of the sea-ports of Spain, and the cases were all among people residing in Madrid who had not been near any infected vessel or any person sick with yellow fever. Thus far everything points to spontaneous origin; but the Spanish physicians, after investigation, arrived at a different conclusion. Associated with the young people who first fell sick, crowded in the same rooms with them to the number of ten or fifteen in a room, were a number of soldiers recently returned from Cuba, *with their baggage.* These men had themselves suffered from yellow fever in Cuba or were acclimated by long residence there; and the outbreak is ascribed by this reporter to "germs" introduced in their baggage. Yellow fever was also epidemic in 1878 at Key West, Fla., 39 deaths; and at Mobile, Ala., 80 deaths.

In the province of Senegal, on the coast of Africa, an epidemic occurred from which resulted a mortality of 650.

1879.—Yellow fever reappeared in Memphis this year; the first case reported to the health office occurring July 8th. At this time the disease was not prevailing in any part of the United States. Dr. *Thornton*, Pres. of the Memphis Board of Health, reports that no communication could be traced between early cases, and says, "The disease appeared in houses in the suburbs, which were infected last year, among people who had no direct communication with the sick this year." Dr. *Coleman* claims to have proof of importation, and Dr. *Thornton* says, "The disease may have been imported this year. If such was the case, the epidemic was in my opinion due to two causes, importation and local origin." Not local origin *de novo*, but as a sequence of the epidemic of last year is evidently what is meant, and there is nothing unreasonable in this supposition, in view of the facts stated as to infected bedding, etc., preserved from the year before; the condition of the privy vaults; the mild climate of Memphis, and the well-attested fact that sporadic cases and minor epidemics have often occurred in New Orleans and other southern cities, not directly traceable to importation. This, indeed, has been the case during the present year, as shown by a very interesting account of a group of cases, commencing in the Stout family (*Holt*). The total number of cases reported at Memphis was 1,532, total deaths, 485. In south-western Louisiana an epidemic, commencing at Morgan City, on Berwick's Bay, resulted in 143 deaths out of a total of 704 cases. Yellow fever prevailed at Morgan City in 1878, and the first case this year (July 25th) was in a locality on the margin of the bay, where numerous cases occurred last year (*Watkins*).

The occurrence of two consecutive epidemics at Memphis from a single importation is contrary to the usual rule, but in my view not at all surprising. The exemption which New Orleans and other southern cities have commonly enjoyed from serious consequences following the appearance of sporadic cases is probably not due to the fact that the yellow fever poison has lost its potency, but to a combination of circumstances, of which the following are the most important. Sporadic cases are usually not developed until late in the season, whereas great epidemics commonly result from an early importation. (In 1878, as early as May 23d.) After a great epidemic, sanitary measures are enforced more rigorously; the doctors, from recent experience, are on the alert and recognize early cases; the health authorities resort promptly to disinfection and other sanitary measures; and, finally, the material is not abundant, as unprotected persons avoid the city which has so recently been scourged by a devastating pestilence.

The facts thus far detailed seem to justify the following conclusions of the Board of Experts appointed by Congress to investigate the epidemic of 1878:

"(29) Yellow fever is transmitted across seas and oceans in steamships and sailing vessels; and the infection may be connected with the vessels themselves, or with the cargoes, or with the crews and passengers."

"(30) Yellow fever is transmitted through the interior of a country by steamboats, barges, and other river craft; and by railroad cars, wagons, carriages, and other land vehicles; and the infection may be attached to the boats, cars, or other vehicles themselves, or to their cargoes, or to the persons travelling upon them, with their baggage."

"(31) The most frequent agency in the dissemination of yellow fever from place to place is found in yellow fever patients; and more epidemics have resulted from the introduction into previously exempt places of persons sick of the disease or falling sick after arrival than from all other causes. To what extent the body of the sick person is responsible for this result, and to what extent his clothing and baggage are responsible for it, is not known."

The admission made in the latter part of proposition thirty-one, that we do not know to what extent the body of the sick person is responsible for the fatal results which often follow his coming to a previously healthy place, is made in a truly scientific spirit, and may be favorably contrasted with the positive assertions for and against personal contagion with which the literature of yellow fever abounds. There are doubtless many facts upon record which seem to point strongly to personal contagion, and one recent writer of prominence (*Bérenger-Feraud*) considers the question settled in the affirmative. The late literature of the subject also indicates that this view has gained ground among the Brazilian doctors (*Rey*). The weight of opinion and of evidence is, however, decidedly upon the other side, and the writer may be permitted to say that in his personal experience, in three minor epidemics, no facts have come under his observation which give the least support to a belief in personal contagion. The following quotation from an article by Dr. *Lawson*, of the English army, is believed to be true as to medical opinion upon this subject:

> "It is worthy of observation that the great majority of the members of the profession who have resided some years in the tropics and had constant experience of yellow fever, entertain the first opinion (non-contagion), and it is only among those who have met the disease occasionally, or who have never been brought in contact with it, that the second (contagion) is generally received."

A few years ago, the only alternative to a belief in personal contagion was the assumption of spontaneous origin *in situ*, at places where the disease prevailed. That such spontaneous origin never occurs it would be impossible to prove, and it must be admitted that the power or combination of circumstances which produced the yellow fever poison in the first instance may produce it again. If, however, as appears probable in the light of modern science, the specific diseases are produced and modified in accordance with the natural laws which govern *evolution ;* then, according to all analogy and the law of probabilities, the independent origin at different times of diseases having the same characters must be very unusual. A few of the striking instances of non-contagion in recent epidemics are the following: Dr. *Ford*, a believer in contagion, reports thirty-five cases of yellow fever among refugees who sought a home in St. Louis during the epidemic of 1878. "In only three of these was any propagation observable." Dr. *Minor* reports that during the same year over thirty cases were discovered among refugees in Cincinnati, Ohio. "No physician or nurse contracted the disease, and in no instance did it exhibit any tendency to spread." In Nashville, during the same year, twenty imported cases occurred in different parts of the city without any local cases resulting from them (Rep. Nashville B. of H.). At Huntsville, Ala., many refugees from Memphis fell sick, but no cases of local origin occurred (*Dement*). In the report of Col. *Cameron*, on the camps established near Memphis, in 1878–79, are the following statements:

> "It was found necessary that the officer in authority should set an example of indifference to attack, in order to appease, as far as possible, the constant

anxiety of the population under his charge. Especially was this true in 1878, as depopulation went on slowly that year, and infected people daily poured into the camps from the more pestilential portions of the city. Very many reached camp with the fever on them, so that as many as seventeen persons fell victims in one night, not a few in their tents. *In no instance, however, did they communicate the disease to their families or bed-fellows*, as far as could be traced. The population of Camp Williams in 1878 was, maximum, 650 souls."

1879. "Average number of people drawing rations from Camp Marks, 1,304; number of yellow fever cases fatal, 9. . . . The general health of the camps was good."

On the other hand, very many instances are on record in which epidemics have resulted without the importation of a case of yellow fever after the arrival of an infected vessel, or the introduction of fomites. In these instances, the first cases sometimes occur from direct exposure, upon opening trunks, handling goods, or visiting an infected ship, and after a short period of incubation, two to five days. But for the development of a new centre of infection a longer time seems to be necessary, and this time varies, according as the local conditions are favorable, from a few days to several weeks or even months. It is this interval required for development and the recognized necessity for certain favorable local conditions, which has recently led so many physicians to look with favor upon the "germ theory," which seems to be the only hypothesis which has been offered capable of harmonizing the apparently contradictory facts with which the literature of yellow fever abounds.

The infection of the bedding of yellow fever patients, and the exceptional cases in which direct personal contagion has apparently been proved, would be explained in accordance with this theory by supposing that the heat, moisture, and organic emanations from the body of a yellow fever patient furnish the local conditions essential for the rapid multiplication of yellow fever germs, and *if these have been introduced from the infected locality in which the patient contracted the disease, infection of his bedding will occur;* or, if germs effect a lodgment in the woollen underclothing of a sailor or laborer, and these garments saturated with moisture and organic matter from his body are worn continuously, the disease might probably be communicated by contagion, *whether the man were sick with yellow fever or proof against it.* It must be admitted that in yellow fever, as in typhoid fever and in cholera, the germ theory has as yet received no support from the numerous attempts which have been made to determine the nature of the poison in these diseases by the use of the microscope and by the various experimental methods known to science. Recent investigations show quite conclusively that there are no microphytes in the blood of yellow fever patients discernible by the highest powers of the microscope as perfected at the present day (Prelim. Rep. of Hav. Com.). But it is in the atmosphere of infected places rather than in the blood or excretions of the sick that we should expect to find a poison which depends for its increase on conditions external to the human body. This is a line of inquiry which has been undertaken by numerous observers, especially with reference to cholera (*Ehrenberg*, *Swayne, Britton, Robin, Thompson, Cunningham,* and others); and recently under the auspices of the Nat. Board of Health, by the Havana Commission in Cuba. The want of success attending all these efforts up to the present time is not evidence of the impossibility of the undertaking, but of its great difficulty. Enough has been done to show that no easy discoveries are awaiting the amateur microscopist in this field. Special training and familiarity with the innumerable harmless microdemes which abound in the atmosphere everywhere, especially in warm climates during the summer months, is essential to the intelligent study of an infected atmosphere by modern scientific methods. The hypothesis that *something given off from the body of the sick, after a time, and with the concurrence of favorable external conditions, becomes, or produces, the true poison of the disease,* seems to be the only alternative of that already given, viz.: A germ multiplying entirely independently of and external to the human body. This would bring yellow fever in the class of miasmatic contagious diseases (*Liebermeister*), with cholera, typhoid fever, etc. This hypothesis does not exclude the idea of a *contagium vivum,* and it comprehends the possibility of the *something* given off from the body being a bioplast (*Beale*), or dead organic matter, capable of changing decomposing substances with which it comes in contact into the efficient cause of the disease, in accordance with the views of *Liebig.*

Of the local causes which favor the increase of the yellow fever poison and consequent extension of epidemics, heat is the only factor which is universally admitted to be essential; but the evidence that atmospheric moisture and the presence of decomposing organic matters of *animal* origin are exceedingly favorable, if not essential, to this increase, has accumulated to such an extent as to appear conclusive on this point. The meteorological researches of *Ford* show an unusually high temperature throughout the region where yellow fever prevailed in 1878, which doubtless had an important bearing upon the rapid spread of the disease.

As to moisture, it seems probable that a sufficient amount is always present in the atmosphere, at those places in the United States where yellow fever is most often epidemic, to admit of the rapid increase of the poison when other conditions are favorable. In Havana, the occurrence of heavy rains has been noticed to be followed by a diminished number of cases, probably because the streets, sewers, and foul places generally, are cleansed by the torrents of water which are discharged through them during a tropical rain-storm.

The influence of filth as a factor is emphatically denied by many observers. The writer is inclined to believe, however, that the necessary filth element might often be brought to light, if a skilled sanitary inspector were to investigate the localities where this element is said to have been wanting; and that the fact that yellow fever did not establish itself in 1878, at Huntsville, Ala., Cincinnati, Ohio, Nashville, Tenn., and at other places where refugees from Memphis and elsewhere sought a temporary home, was in great part due to the superior hygienic condition of these towns in this particular. At Fort Barrancas, Fla., in 1875, nothing could be found in the police of the garrison or its immediate surroundings to which exception could be taken. But upon the sea-beach, a mile away, and in the direction of the prevailing winds, was an accumulation of seaweed thrown up by an unusually severe storm from the south-west. A very offensive odor from this direction, especially at night, indicated that this mass of seaweed and the shell-fish, medusæ, etc., mingled with it, was undergoing putrefactive changes. It may be that the algæ and fungi are an exception to the general rule that decomposing vegetable matters do not furnish a favorable nidus for the development of the yellow fever poison. Whether this is the case or not, there is always upon the sea-beach, and especially adherent to and mingled with stranded algæ, a considerable amount of animal matter; and many facts might be given indicating that this may become an important factor in the production of an epidemic of yellow fever.

The limited space alotted to this article forbids a more extended discussion of etiological questions, nor would such discussion throw any additional light upon the interesting question of the true nature of the yellow fever poison. As to *its specific character; its exotic origin; its frequent importation to our shores from places where the disease is endemic; and the fact that it requires heat, moisture, and decomposing animal matter for its development*—all of these points seem to be established with sufficient certainty to furnish a basis for intelligent measures of prophylaxis.

YELLOW FEVER OUTSIDE OF THE UNITED STATES.

The continued prevalence of yellow fever, or its endemicity, in Havana, is shown by the following tables extracted from the Preliminary Report of the Havana Commission.

Monthly maximum and minimum deaths by yellow fever in Havana, during the ten years, 1870–9.

Months.	Minimum.	Maximum.
January	6	32
February	4	24
March	4	32
April	4	37
May	13	103
June	66	378
July	68	675
August	70	416
September	35	234
October	28	185
November	5	150
December	9	82

In no one of the ten years, 1870–1879, have there ever been fewer deaths than in the first, nor more than in the second column, and the total deaths by yellow fever for each year were as follows:

Total deaths by yellow fever in Havana.

In 1870	665
In 1871	991
In 1872	515
In 1873	1,244
In 1874	1,425
In 1875	1,001
In 1876	1,619
In 1877	1,374
In 1878	1,559
In 1879, to October 1st	1,353

The continued prevalence of the disease at Rio de Janeiro, for ten years is shown by the following table, extracted from the National Board of Health Bulletin.

The table was prepared by J. J. C. Voigt, public translator, and its accuracy is vouched for by the United States consul-general, Thomas Adamson.

Fatal Cases of Yellow Fever.

		January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
1869		....	..	....	....	....	....	....	....	...	....	....	....	274
1870		....	....	....	...	....	....	....	...	....	....	....	....	1117
1871		....	....	....	....	....	....	....	....	....	....	....	...	8
1872		1	....	..	....	1	3	1	. ..	1	8	16	71	102
1873	1st fortnight	262	582	592	162	96	51	9	....	1	5	....	7	3467
	2d fortnight	627	505	316	113	84	32	11	3	1	....	2	6	
1874	1st fortnight	4	13	65	161	97	38	11	5	....	2	4	7	826
	2d fortnight	12	38	103	136	68	31	14	....	1	4	6	6	
1875	1st fortnight	1	61	196	159	127	74	26	7	5	1	3	....	1292
	2d fortnight	22	107	189	142	117	30	11	3	....	3	....	8	
1876	1st fortnight	46	109	536	597	220	87	28	6	1	1	2	1	3317
	2d fortnight	76	210	821	349	155	42	13	6	5	3	2	1	
1877	1st fortnight	1	....	25	12	15	7	3	10	2	4	8	19	213
	2d fortnight	2	10	14	11	7	4	1	5	5	4	10	34	
1878	1st fortnight	41	164	142	65	32	6	12	3	3	5	1	6	880
	2d fortnight	36	162	122	34	10	13	6	1	5	5	1	5	

"Vera Cruz, Mexico, has been for at least two centuries a continual centre of infection," sporadic cases constantly occur, in winter as well as in summer. From this city it is taken to other places on the Mexican coast. It is endemic at Alvarado, eighteen leagues from Vera Cruz, and at Campeachy. Between Alvarado and Campeachy are several ports (twelve) which have a considerable commerce in precious woods. These places do not suffer in the least from yellow fever, ships come to these ports in ballast, *not having touched at any port where yellow fever exists;* the crews, Swedes, Norwegians, and English, work hard in the hot sun and suffer from malarial fevers, but never from yellow fever (*Heinemann*). Yellow fever is endemic at Maracaibo, Venezuela (*Dagnino*).

Bérenger-Feraud gives the following interesting historical statement of the prevalence of yellow fever upon the island of Martinique.

> The disease prevailed annually from 1682 to 1708, 26 years. The island then enjoyed a complete immunity for 11 years; followed by 16 years "epidemic;" 13 years immunity; 7 years prevalence; 6 years immunity; 12 years prevalence; 17 years immunity; 18 years prevalence; 7 years immunity; 14 years prevalence; 8 years immunity; 7 years prevalence; 6 years immunity; 7 years prevalence; 11 years immunity; and, finally, annual prevalence from 1869 to 1877.

At Martinique, as in all places where the disease is endemic, cases occur during every month in the year.

PROPHYLAXIS.

Quarantine, Sanitation, Depopulation of Infected Places.

This is the tripod upon which preventive medicine must rest; and, in view of the acknowledged impotency of our therapeutics, the importance of these measures, and their demonstrated success in saving life, cannot be too strongly insisted upon.

A full discussion of the complicated questions connected with the execution of these measures would be out of place in this article; but the following axioms, based upon our present knowledge of the disease, are offered as safe rules for guidance in prophylaxis.

Quarantine.—A time-quarantine, and reliance upon the sickness of passengers or crew, as a test of the infection of a vessel is unsafe, and unjustifiable in the light of present knowledge and past experience.

A vessel from an infected port, during the yellow fever season, should always be considered dangerous, and detention at quarantine for a longer time than is necessary for the cleansing and disinfection of a vessel adds to this danger.

Ballast from infected ports is dangerous.

The thorough application of the most approved methods of cleansing and disinfection cannot always be relied upon for ridding a ship of the infection of yellow fever; but repeated disinfection, cleanliness, and free ventilation reduce the danger to a minimum.

The practice of these measures at the point of departure and during the voyage would, probably, be of the greatest utility.

Sanitation.—It is safer, and cheaper in the end, to prevent accumulations of filth than to disinfect them.

For this purpose, properly constructed sewers and an ample supply of water are of the highest importance. Disinfection should always be considered secondary to cleanliness and free ventilation.

In disinfection, great results must not be expected from a small outlay of labor and material.

Quarantine and sanitation cost less than epidemics.

The loss to commerce from non-intercourse with infected ports during the dangerous months would in many years amount to less than the loss to the country by a single epidemic like that of 1878.

Depopulation.—No unprotected person should remain in an infected locality unless compelled to do so by duty or urgent necessity.

The prompt removal of all unprotected persons from infected ships, or towns, is the evident duty of those who have the control of measures for the protection of the public health.

Personal Prophylaxis.—Fear predisposes to an attack, but the absence of fear furnishes no protection, as has been proved by many misguided persons at the cost of their lives.

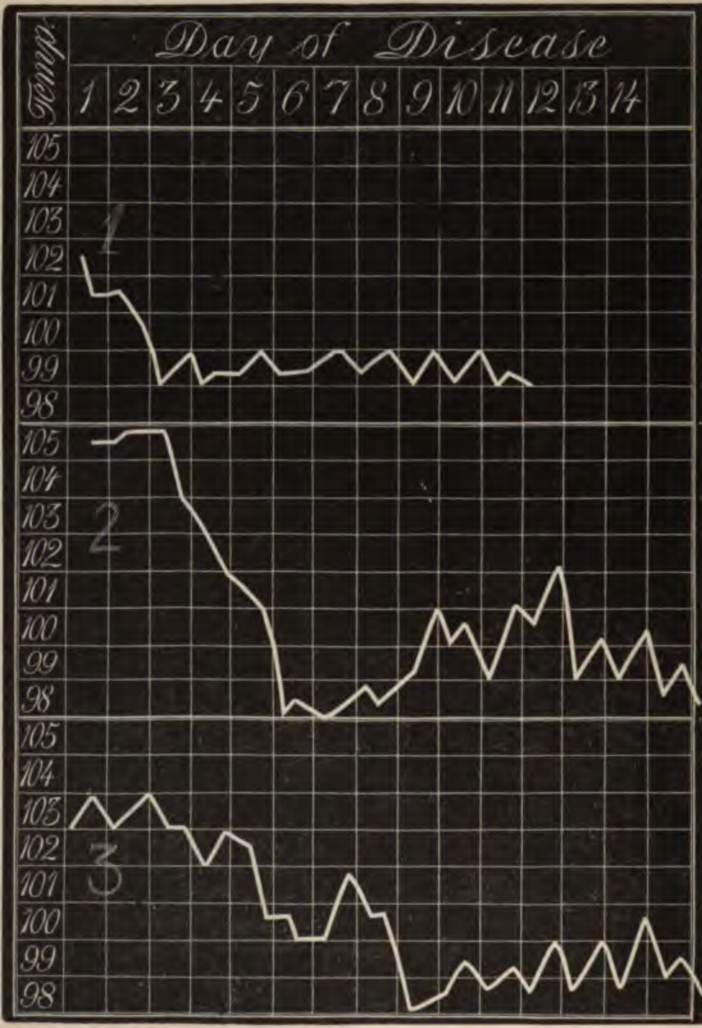
A reliance upon certain drugs, or precautions as to mode of life, has also proved fatal to many persons who have been led by business or pleasure to visit infected localities. No such preventive measures are known to science, and the chances of death taken by such venturesome persons are greater than if they were going into battle.

For persons detained in an infected city by duty or necessity the best advice that can be given is to maintain a cheerful frame of mind; avoid excesses of all kinds; keep away from centres of infection (as shown by the occurrence of numerous cases); sleep as far from the ground as possible; *keep the bowels open.* In malarial localities it is well to take prophylactic doses of quinine, as an attack of malarial fever, like anything else that disturbs the balance of health, is very likely to eventuate in an attack of the prevailing disease.

CLINICAL HISTORY.

Yellow fever, as it has occurred in the United States during the period covered by our historical summary, does not differ from the disease as described in standard medical works published prior to this period. But the numerous careful observations with the clinical thermometer which have been made during recent epidemics have aided greatly in defining the specific character of the disease, and have made diagnosis and prognosis more easy and certain, especially for the inexperienced. These observations have demonstrated the fact that *yellow fever is a continued fever of a single paroxysm of indefinite duration, but having a tendency to terminate in two or four days, or a multiple thereof.* It is only by a study of non-fatal and comparatively mild cases that this tendency to a definite duration can be observed, and it must be admitted that even in these there are many exceptions to the rule. The duration of the characteristic paroxysm or initial fever only is referred to in speaking of the duration of yellow fever; but the termination of the case is by no means reached when the initial fever, which is doubtless produced by the direct effects of the poison, has run its course. On the contrary, the period of apyrexia which follows, of from a few hours to two or three days duration, is an extremely critical stage of the disease, during which the question is to be decided as to whether the powers of nature are equal to the repair of damages produced during the paroxysm. In favorable cases a reactionary fever follows this period of apyrexia, which is of irregular course and uncertain duration. In very mild cases it may be absent entirely, or so slight as to escape observation; indeed, in the majority of temperature charts which I have examined, it is not shown, as observations are commonly discontinued when the temperature has once reached the normal.

The following charts are given as representing typical cases of two, four, and eight days duration. In No. 2 the attack commenced on the evening of the first day, and a complete remission was first observed on the morning of the sixth day, but may have occurred during the preceding night, in which case the duration of the paroxysm would be exactly four days; and if this was not the case, it was but a few hours more as shown in the chart.



Remarks.

Male, aged 26; Barrancas, Fla., 1873. Typical mild case of 48 hours' duration; reactionary fever slight.

Male, aged 27; Barrancas, Fla., 1873. Typical severe case of four days' duration; three stages well-marked; initial fever four days; stage of calm three days; reactionary fever six days.

Male, aged 27; Barrancas, Fla., 1874. Mild case of eight days' duration; brief stage of calm; slight reactionary fever.

In a paper published in 1875, the writer, from an analysis of 140 cases, arrived at the conclusion that the above charts represent three distinct types of the disease, designated respectively, "*simple, duplex, and quadruplex.*" Subsequent observations have shown that so large a proportion of cases fail to conform to any of these types that the value of the classification may be questioned. In many cases it happens that the temperature remains one or two degrees above the normal during the stage of calm, and there is, consequently, no well defined dividing line between the initial and the reactionary fever. Complications (visceral congestions, abscesses, parotitis, etc.), indiscretions in diet, active medication, and moral causes (especially fright and grief), all influence the temperature in a marked manner and reduce materially the number of cases in which the typical course is maintained.

The most striking peculiarity of the temperature curve in yellow fever is the fact that the highest point is reached at the outset of the disease, and that, as a rule, after the acme of temperature is reached, defervescence occurs by a continuous fall to the normal, or even a degree or two below. In mild cases, the acme is reached during the first two or three hours of the attack. In more protracted and severe cases the acme is not reached until the second or third day, rarely later. In 192 cases recorded by *Faget, Jones*, and myself, the acme was reached on the first day in 102, on the second in 54, on the third in 33, and on the fourth in 3. Jones records a temperature of 110° in no less than four cases, and in several others 109° and 108°. In *Faget's* tables the highest temperature recorded is 107.2°.

Thornton notes a temperature of 108° in a single instance, at Memphis, in a total of 143 cases. With this exception, 106½° is the highest temperature recorded by him. In my own observations, 106° has been the highest temperature noted.

The course of yellow fever is doubtless modified in many cases by a malarial complication, and in certain epidemics occurring in highly malarial regions this element is so marked as to give a distinctive character to the compound disease and to call for special treatment. The epidemic in Savannah in 1876 is said to have been of this character; and one author (*Waring*) makes the statement that for one case of yellow fever there occurred a hundred cases of malarial intermittent and remittent.

Bérenger-Feraud admits that "*paludisme*" may complicate yellow fever in two different manners. On the one hand, a person suffering from malarial poisoning, being attacked by yellow fever, "may suffer accidents more or less grave" from this complication; on the other, the convalescent from yellow fever in his condition of profound anæmia is more subject than another to the influence of the paludal poison.

In certain undoubted cases of yellow fever, the paroxysm is divided by a more or less complete remission into two or more distinct periods, each of two or four days duration. Attention was called to this fact, by the writer, in 1875. The number of recorded cases, however, which present this peculiarity is not sufficient to justify a modification of the general s[illegible]ment that *yellow fever is a continued fever of a single paroxysm.* [illegible]re [illegible]y be that these are hybrid cases and that the peculiarity in t[illegible]rature curve is due to the malarial element in the case. This p[illegible]ty is well shown in two charts, given by *Primet*, of cases occurring in French Guiana.

Bérenger-Feraud, whose opportunities for the study of yellow fever, both on the coast of Africa and in the French Antilles, have been unsurpassed, and who has contributed largely to the recent literature of the disease, describes it as occurring under two forms and of four degrees of intensity, viz., *forme franche et forme insidieuse; degré léger, degré moyen, degré intense et degré sidérant.*

This author also devotes a volume to the description of a fever known in the Antilles and in tropical America under the following names: *Fièvre inflammatoire, gastro-céphalite, gastro-cérébral; fièvre d'acclimation, rémittente bilieuse; fièvre jaune bénigne, fièvre jaune abortive; fièvre jaune des creoles; dengue, etc.*" He says: "These fevers may exist sporadically, like the yellow fever, and also epidemically, but *it is above all at the approach or decline of the epidemics of yellow fever that they are observed in the greatest number*. They present different forms; the most frequent form is observed among people who are subject to be attacked by yellow fever. It offers then all the symptomatic appearances of the first degree of yellow fever—coloration of the skin and eyes, elevation of temperature and pulse, cephalalgia, rachalgia, contusive pains in the limbs—but whatever may be the intensity of these symptoms, they all disappear at the end of twenty-four or forty-eight hours and recovery takes place." In 1875 *Bérenger-Feraud* lost but three cases in 400, and in 1876 one case in 210.

Such is the resemblance of this form of fever with the first degree of yellow fever that when it is observed sporadically without an epidemic of yellow fever, the doctors of the country say: "If we were in the time of yellow fever we would say that it is yellow fever." *Bérenger-Feraud* claims that this fever prevails everywhere that yellow fever reigns; and says: "It is a disease very near, if not identical with yellow fever—an incomplete yellow fever." At St. Pierre, where this fever reigns, malarial fevers do not prevail. Quinine is of no use in the treatment of these fevers.

The prognostic value of temperature observations in yellow fever is shown by the following table:

Cases in which the temperature was	No. of Cases.	No. of Deaths.	Percentage of Deaths to Cases.
107° and above	13	13	100
106° — 107°	9	9	100
105° — 106°	36	22	61
104° — 105°	80	24	30
103° — 104°	87	6	7 nearly
102° — 103°	29	0	...
101° — 102°	15	0	...
Total....	269	74	27.5

The temperature curve considered in connection with [illegible]e pulse, as pointed out by *Faget*, is probably the most reliable guid[illegible] have in diagnosis. The pulse is most rapid at the very outset of th[illegible] and a graphic representation of its frequency is usually a descen[illegible]line which is parallel with the temperature line in those cases where this falls at once. In cases in which a high temperature is maintained for two or three days, the pulse also falls from the outset, so that the two lines do not become parallel until defervescence commences. A considerable rise of temperature shortly before death occurs in a certain number of cases, and several instances are recorded in which a post-mortem elevation of temperature took place.

The most thorough, recent, chemical study of the excretions is that of *Cunisset*. This author finds that the urine of yellow fever contains less than the normal amount of urea. In mild cases this difference is scarcely appreciable, but in severe cases it is reduced to an extremely small amount. When the amount increases it is a favorable symptom. Uric acid has also appeared to diminish in amount, but in much less proportion than the urea. "We have seen urine containing only seven grammes of urea per litre give a relatively abundant deposit of uric acid." Biliary pigments usually appear in the last days of sickness, and when the urine is abundant their presence is rather a favorable prognostic sign.

Black vomit was sometimes found to contain a small amount of urea, but never carbonate of ammonia, which indeed could not exist in the acid black vomit, nor was carbonate of ammonia found in the fæces. It cannot, therefore, be admitted that the urea, not being eliminated by the kidneys, is discharged by the intestines. Nor is urea found in appreciable quantity in the serosity of blisters; an analysis of which enables the author also to affirm that the icteric tint is due to "*hémaphéine*" and not to the pigments of the bile. The diminished excretion of urea found by *Cunisset* is confirmatory of observations previously made by *Lawson* and others, but is contradicted by the analysis of *Jones*, who says: "Where there is no suppression of the urinary excretion, the urea is increased above the standard of health, during the active stages of the disease and during the period of exhaustion or calm."

In 1875, the writer made a series of observations upon the amount and specific gravity of urine excreted by yellow fever patients with a view to determining *what should be the amount of urinary salts excreted each day during the favorable progress of a non-fatal case of yellow fever.*

No analysis was attempted, but the total amount of urine passed by each patient was carefully measured, and the specific gravity was taken twice daily. The amount passed during twenty-four hours, considered in connection with the specific gravity, is, of course, a true index of the total solids excreted. As 1,000, the specific gravity of water, is a constant factor in such an estimate, it may be eliminated. It is estimated by physiologists (vide *Flint's* Physiology, Vol. Secretion, p. 189) that a healthy adult should secrete fifty fluid-ounces of urine having a specific gravity of 1,020, during the twenty-four hours. Eliminating 1,000 from the second factor, we have (50×20 =1,000) one thousand as the index of total solids excreted in twenty-four hours. Taking the average of sixteen non-fatal cases of yellow fever, I obtained the following results:

	Amount in fluid-ounces.	× S. G. — 1000	
Normal in adult male....	50	× 20 =	1000
1st day of yellow fever	8	× 23 =	184
2d “ “ “	11.5	× 25 =	287
3d “ “ “	16	× 28 =	448
4th “ “ “	18	× 22 =	396
5th “ “ “	19	× 22 =	418
6th “ “ “	20	× 22 =	440
7th “ “ “	22	× 21 =	462
8th “ “ “	22	× 19 =	418
9th “ “ “	23	× 16 =	368
10th “ “ “	28	× 13 =	364
11th “ “ “	37	× 11 =	407
12th “ “ “	41	× 13 =	533

It will be seen that the product of the amount multiplied by the specific gravity is tolerably uniform from the third to the twelfth day, and that this uniformity is preserved by a daily falling off of the specific gravity to compensate for the daily increase in quantity. To ascertain the product of amount multiplied by specific gravity in patients convalescent from yellow fever at a later date, I had the urine passed by sixteen convalescents, on full diet, who had been out of bed from ten to twenty days, preserved and measured. The result was, that the average of amount multiplied by the specific gravity for the sixteen cases was 491—a product but little in excess of that obtained during the continuance of the fever when the patients were quiet in bed. The figures in the table may then be taken as representing, approximately, the normal quantity of urinary solids which should be excreted daily during the progress of an attack of yellow fever; and if the amount falls materially below these figures, defective excretion may be premised, and treatment and prognosis governed accordingly.

PATHOLOGY.

The chief interest in recent contributions to the pathology of yellow fever is attached to the attempts which have been made to establish a pathological histology of the disease; and in the microscopic examination of the blood which has engaged the attention of several observers. The writer gave especial attention to the latter study while in Havana in 1879, and examined a large number of specimens from patients in every stage of the disease. A *Zeiss'* $\frac{1}{18}$ inch objective failed to show any microphytes; and the disorganization of the blood so often spoken of as characteristic of yellow fever was found not to exist so far as the cellular elements are concerned, even in blood taken a few hours before death. These observations are confirmed by *Heinemann*, who examined the blood of patients in the last stage of the disease, at Vera Cruz, taking blood from the hand, thinning it with artificial serum, and bringing it at once under the microscope. He says "in nine cases not the slightest deviation from normal blood could be found; nothing of the destruction beginning in the red corpuscles. . . . The white corpuscles, so far as could be estimated, were not increased in number. . . . No organisms were found."

According to my observations, in cases near a fatal termination, the number of white corpuscles is reduced, and they contain an unusual number of fat granules, which I have attributed to a fatty degeneration of the protoplasm, but which may have been picked up in the blood, suspended in which are seen, in certain cases, a considerable number of free fat globules, extremely minute. The serum of blood drawn near the close of a fatal case, and of post-mortem blood, soon becomes deeply colored from a solution of the coloring matter of the red corpuscles, and sometimes numerous crystals of hæmatoidine are formed in blood which has been kept for a considerable time. This solution of the coloring matter probably takes place to some extent during the progress of the disease, and no doubt the yellow coloration of the skin from which the disease takes its name is due to this fact. The fibrinous element is diminished in quantity, but not entirely absent, as has been claimed. In specimens of post-mortem blood sent to me from Havana, I have found crystals of ammonio-magnesian phosphate, and in blood kept for a considerable time in culture-cells stellate acicular crystals are sometimes formed, the nature of which I have not determined. Finally it may be said that observations already made indicate than any changes which may occur in the circulating fluid are to be sought by the chemist rather than by the microscopist. The following are the conclusions of *Cunisset*, whose chemical study of yellow fever is the most recent, and apparently the most thorough, which has been made.

"Yellow fever is not a poisoning by the bile ; at the outset of the malady the biliary pigments are rarely found in the blood or in the urine. They appear generally only during the second period, and in a great number of cases they are not to be found at all.

"The biliary salts, of which the powerful action of 'deglobulization' admitted by certain authors might explain the disorders which the malady presents, do not exist either in the matters vomited, or in the urine, or in the blood, except, in certain cases, in very feeble quantity. In view of the profound alterations of the liver, this absence of the biliary salts is easily understood, and the defective depuration of the blood is to be looked upon as a complication rather than a determining cause of the malady."

"Yellow fever is not a poisoning by cholesterine, as this substance is not found except in quantities scarcely appreciable. The fæces do not contain stercorine, the absence of which proves that the blood does not contain an abnormal quantity of cholesterine.

"Yellow fever is not a poisoning by urea. In place of an accumulation of this substance in the blood, there is rather a diminished production of urea. The liver, the chief agent in the formation of urea, is invaded by fat, its functions are diminished, if not completely abolished; urea is, therefore, necessarily diminished in quantity. In cases complicated by suppression of urine, death arrives rapidly, not because urea is not eliminated, but because the blood no longer finds an outlet for the expulsion of the products of incomplete disassimilation intermediate between urea and the albuminoid substances which it contains."

If this view is correct, may not the albumen in the urine be evidence of an effort of nature to depurate the blood, through the kidneys, of injurious albuminoid substances? May not the structural changes and imperfect action of the kidneys result to some extent from this unnatural duty which they are called upon to perform, instead of the albumen appearing in the urine as a result of imperfect action and structural change in the kidneys?

"Yellow fever is not a poisoning by carbonate of ammonia. The blood does not contain carbonate of ammonia. This salt is very easily eliminated by the kidneys and if present would neutralize the acidity of the urine."

"What then is yellow fever? In yellow fever, in the first line, we will place the fatty degeneration of the glandular and muscular tissues and the passage of the hæmoglobine into the serum. For us, these two facts are joined the one to the other; they permit us to give an explanation of the malady."

But what is the cause of this fatty degeneration? Recent experiments by *Cunningham*, of the English army (*Quar. J. of Mic. Sc.*, N. S., No. LXXVII., p. 50), show that starvation of animal and vegetable tissues gives rise to a fatty degeneration of their protoplasm.

Starvation of animal tissues may arise from two causes: (a) deficient supply of nutriment; (b) total abolition or diminished activity of the controlling power exercised through the nervous system upon processes of nutrition. The first cause may evidently be excluded in yellow fever, as seizures occur when the blood is still loaded with the digested products of a recent meal.

As to the second; all the early phenomena of the disease point to a profound impression made upon the nervous system by the specific poison of yellow fever. The suddenly developed high temperature, the cephalalgia, the rachalgia, the flushed face, the injected conjunctivæ, the hyperæmia of visible mucous membranes, the congestion of the viscera; in short, the passive dilatation of the capillaries throughout the body, is evidence of vaso-motor paralysis; producing on a large scale the effects which section of the sympathetic nerves going to a particular part produces in the limited area, supplied by the divided nerves (vide *Flint's* Physiology, Vol. Secretion, etc., p. 416). This general hyperæmia, is, so far as we know, the first effect of the yellow fever poison; and any previous changes in the blood, resulting from a process of zymosis, are entirely hypothetical. If death occurs within a few hours, or a day or two after the seizure, the pathological phenomena are those of congestion. The liver is dark-colored and gorged with blood; the brain and its meninges, the kidneys, the mucous membranes, all present evidences of hyperæmia. Later occur the fatty degeneration and the blood changes which are characteristic pathological features of a case in which death occurs after the second or third day; and which, in our view, are secondary phenomena resulting from the arrest of vital processes—nutrition, secretion, excretion—presided over by the nervous system.

This view of the *modus operandi* of the yellow fever poison is supported by a variety of facts. Observation indicates that the severity of an attack bears a direct relation, other things being equal, to the amount of the poison taken ; and that a tolerance—acclimation—is acquired by repeated exposure to its influence. This has its analogy in the effects of the known poisons which act upon the nervous system—*e*, *g.*, opium, tobacco. In a true blood-poison the effects are immediate and may recur any number of times. The interval of from twelve hours to four or five days which elapses after exposure before the attack occurs does not seem to be a true period of incubation, such as we have in small-pox and measles; and the development of an attack after exposure may probably be, sometimes, postponed or prevented altogether by avoiding excitement and fatigue, and keeping the bowels open. On the other hand, excessive fatigue, mental anxiety, venereal indulgence, and, in general, those causes which have a depressing influence upon the nervous system, may hasten the development of an attack, or produce it in those who are little susceptible to the influence of the poison. The delay—so-called period of incubation—which occurs after exposure, before the phenomena of the disease manifest themselves, has its analogy in the action of certain known poisons. In poisoning by mushrooms the symptoms, sometimes, do not occur until thirty hours after the meal is taken.

Finally, recent pathological observations indicate that the nervous system is seriously implicated in yellow fever. *H. D. Schmidt,* who has made a large number of autopsies, giving especial attention to the condition of the nervous system, reports the brain congested, sometimes throughout, sometimes in certain portions, especially the parietal lobe. During the last epidemic (1878), he examined the spinal narrow and the sympathetic system, especially the semilunar, and the first thoracic sympathetic ganglia. To his surprise he found that in the semilunar ganglia, and also in the thoracic, the nuclei of the ganglion cells were entirely gone and besides that the ganglion cells had a true fatty lustre. In 551 autopsies made by the French physicians at Martinique, the brain was examined 473 times and the following conditions noted: congestion of the meninges; increased amount of fluid in the ventricles and arachnoid; small hemorrhagic extravasions—"*petites piquetés hemorrhagiques*"—in the brain and softening of its substance.

The histological examinations made by the Brasilian physicians at Rio and Bahia in 1873, 1875, and 1876 showed that the gray substance had undergone, in certain points, a fatty degeneration, while the white substance presented nothing abnormal (*Bérenger-Feraud*).

The pathological histology of yellow fever has recently occupied the attention of numerous observers; but, in some respects, their results are contradictory and additional observations will be required to establish this interesting branch of pathology upon a firm basis.

Heart.—"The examination of this organ shows that there is no foundation for the opinion that there is a fatty degeneration of the muscular fibre. The heart is almost always found firmly contracted, and its consistency and color are normal. The striations are always distinct, and only in some of the fibres a few fatty granules are found in the neighborhood of the nuclei" (*Guiteras*). "The heart substance was soft, flabby, and slightly fatty" (*Satterthwaite*). "Fatty degeneration of muscular fibres of the heart and oil globules between the transverse striæ of the fibres (*Gama Lobo*, reported by *Rey*). "The heart is generally empty and very firm. No fatty degeneration of the muscular fibres was found" (*Crevaux*).

Kidneys.—"The epithelium throughout the kidney was swollen and intensely granular. The tubes contained numerous small granular casts of a yellowish color. At the apices of the pyramids, the collecting tubes were nearly stripped of epithelium. The epithelium that remained was intensely swollen, and even more granular than in other parts of the kidney" (*Satterthwaite*). "In forty-one autopsies, lesions of the kidneys were found forty-one times. Until now, authors have not insisted on fatty degeneration of the kidneys. We call particular attention to some pathological manifestations which precede this alteration. . . . There is then, in the kidneys, as in the stomach and in the liver, at first a state of congestion, which may be followed by apoplectic effusions in different parts of these organs. The second state of this process is a fatty degeneration of the cells which form the renal parenchyma" (*Crevaux*).

Liver.—"The topographic study of the sections shows the lobules perfectly distinct; no alteration of the central veins; *no trace of connective tissue proliferation* in the interlobular spaces. . . . The hepatic lobules are either totally degenerated, or present yet some parts of normal appearance. Around the central vein in some lobules, one finds a band (*une colerette*) of healthy tissue. . . . Two modes of alteration have invaded the substance of the lobule: 1st, fatty degeneration of the cells; 2d, granular fragmentation of these same elements. The two alterations are intimately mixed, but the second occupies the greater place (Examination in *Charcot's* laboratory by *Sabourin*). "On microscopic section, the liver was found to be in an advanced stage of fatty degeneration. . . . There was also a marked increase in the connective tissue of the organ" (*Satterthwaite*).

Stomach.—"The whole surface was slightly congested, and overlaid with a soft coating of mucus, which, when brushed off, showed many little minute black points, representing black vomit matter; these little black points seemed to be at the mouths of the follicles. On microscopic examination, all the coats of the stomach appeared a little thickened, but most markedly the mucous or follicular coat. Some of the little points were seen under the microscope. The epithelia of the mucous and peptic glands were thought to be abnormally granular. At the mouth of the follicles the epithelium was frequently wanting" (*Satterthwaite*). "It is very difficult at first to find the source of the blood found in the black vomit. The reason is this, that the tops of the ridges between the gastric follicles are removed by post-mortem digestion, and it appears that the hemorrhages take place always, as far as I have seen, from the loops of capillaries that rise into these ridges. The post-mortem digestion removes, therefore, the hemorrhagic foci that could lead us to detect the points of hemorrhage" (*Guiteras*). "The histology of the mucous membrane of the stomach has revealed profound alterations, even in those cases where this membrane has not appeared very much changed; on the one hand, *a fatty degeneration of the capillary vessels;* on the other, a fatty degeneration of the epithelial cells of the stomach glands. M. Crevaux has found these lesions in all his investigations at the *îles du salut,* and he considers them as pathognomonic of yellow fever, an opinion which we share entirely" (*Bérenger-Feraud*).

TREATMENT.

Recent epidemics, and especially that of 1878, have given many physicians in the United States experience in the treatment of yellow fever, who had previously known of its malignant nature only by report, but the combined efforts of the many to combat this deadly foe has only given additional weight to the conviction, long since reached by the experienced few, that *active medication in yellow fever is positively injurious.* We should not cease to hope that an antidote to the yellow fever poison will yet be found, and any new remedy, or mode of treatment, offering a rational hope of success should be fairly tested; but, in the interest of future patients, it is highly important that this lesson, learned, it is to be feared, at great expense of life, be accepted upon the authority of those who have seen most of the disease. While all efforts heretofore made to *cure* yellow fever have been futile or positively harmful, the writer believes that skill and experience on the part of the medical attendant influence the result in this disease to a degree exceeded in but few, if any, others. It is true that many cases are of so mild a type that recovery may take place under the most adverse circumstances and even in spite of heroic treatment. On the other hand, certain cases are of so malignant a character that no amount of skill and care can avert a fatal termination; but between these extremes is a considerable number of cases in which the balance of life and death is in the hands of the physician. An ill-timed cathartic, a discouraging word, permission to sit up, or to partake of solid food, exposure to draughts, in short, many things which to the inexperienced may appear trivial, when thrown into the balance, in this disease, may turn the scale to the fatal side. On the contrary, encouraging words to the patient, positive and judicious instructions to nurses, frequent visits and vigilant supervision of all that occurs in the sick-room, the timely administration of stimulants, and in short, close attention to all the details of what is known as "*nursing*" will tide many a patient over the critical periods of the disease, and save his life, which, so far as he is concerned, is quite as good as "curing" him.

The limits of this article forbid an extended discussion of the various modes of treatment which have been proposed, and only that which is believed to have given the best results will be here detailed; followed by brief reference to recent suggestions which seem worthy of attention. It is generally admitted that it is desirable to administer a prompt cathartic at the outset of an attack and many of the Spanish and Portuguese physicians give an emeto-cathartic. There is probably no better medicine for unloading the bowels than the standard "creole" dose ot castor oil which may be repeated if necessary, or followed by a soap and water enema. An emetic is seldom needed, but if the seizure is soon after a full meal and there is nausea without vomiting, it is well to aid the efforts of nature by giving an emetic of mustard and warm water or of ipecacuanha. Give a hot mustard foot-bath and put the patient to bed, covering him *lightly* with blankets; promote perspiration by warm drinks, or cold, if preferred—orange-leaf tea, hot lemonade, weak black tea, or, simply iced water. These drinks may be given ad lib. unless the stomach is inclined to reject them, in which case give small quantities, one or two tablespoonfuls, frequently repeated, and apply hot bricks to the feet, and sinapisms to the epigastrium. Keep the patient perfectly quiet in bed, and endeavor to maintain a gentle perspiration, as this will conduce to his comfort and safety; but do not imagine that the disease can be cured by "sweating." A load of blankets and excessive perspiration annoys and weakens the patient. Bedding should be changed, with as little disturbance to the patient as possible, whenever it becomes soiled by discharges or saturated with perspiration, care being taken to protect the patient from draughts, and to warm the fresh blankets. The sick-room should be large and well ventilated, and it is desirable never to have two patients in the same room. The cold bath may be used during the first twenty-four hours, if the temperature exceeds 104° Fah.; but, later, it is doubtful whether the benefit derived from a temporary reduction of temperature would counterbalance the injurious effects of the disturbance of the patient necessarily attending its administration. It is, therefore, preferable to reduce temperature by frequent sponging of the body, under the bed clothes, with water or an evaporating lotion. Baths and sponging are especially to be recommended when the skin is dry and pungent. When the patient is perspiring, keep him lightly covered and await a reduction of temperature as a result of the natural evaporation from the surface.

That quinine is not curative in yellow fever is amply demonstrated; but considerable difference of opinion exists as to the valne of this medicine as an antipyretic. Some writers contend that it is positively injurious (*Bérenger-Feraud*); others, that it is harmless, but inefficient; and others give it in full doses at the outset of the attack. Anything less than thirty grains may be considered too small a quantity to test fairly its antipyretic value.

In favorable cases, no other treatment is required than that above indicated. No food should be given before the third or fourth day, when light liquid nourishment may be cautiously given. Commence with milk and lime-water, or chicken-broth in small quantities, every two or three hours; increasing the dose if the stomach bears it well; if not, reducing it or withholding it entirely for twenty-four or forty-eight hours longer. Rectal alimentation should be resorted to when an irritable stomach prevents the administration of food *per orem*.

The indication for administration of food is not, however, so urgent as the appearance of the patient would indicate. If his adynamic condition is due to starvation, it is not a starvation due to withholding of food, but from the arrest in the glandular cells throughout the body, of processes of absorption, secretion, and excretion which are essential to the nutrition of the tissues, and which, when the circuit is complete, as in the healthy individual, may go on for many days, independently of a new supply of pabulum, without causing any such marked prostration. This is further shown by the remarkable rapidity with which strength is regained, in certain cases, in which a prompt convalescence indicates that the functional activity of the paralyzed cells has been recovered. The glandular cells, in which the most marked pathological lesions are found, seem to furnish no exception to the general rule that rest is the great restorer of enfeebled and diseased organs, and any attempt to force a resumption of their functional activity before nature has had time to repair damages is likely to prove disastrous. Some of the Brazilian physicians withhold food for a much longer time than that above indicated. "Food is not given until all danger is past" (*Waugh*).

Stimulants in small quantities frequently repeated may be commenced on the third or fourth day, or sooner if indicated, *and desired by the patient*. In certain cases their liberal use will be required until convalescence is fully established. At first iced champagne, in tablespoonful, or good brandy, in teaspoonful doses; repeated every hour, or oftener, will be the best form. Later, milk-punch, English ale or porter, may be given in liberal quantities, especially to those who are in the habit of using spirits. On the fifth day, or later, when the patient's stomach has behaved well, easily digested solid food may be cautiously added to his bill of fare. Indigestible food, or excessive indulgence, must be guarded against until all danger of a relapse is past, by three weeks at least.

The various distressing symptoms and complications are to be combated by external applications and hypodermic medication so far as possible.

Headache is best relieved by cold applications to the head and sinapisms to the extremities; epigastric distress and nausea by small quantities of carbonic acid water, lime-water, or sodæ bicarb., and by the application of sinapisms to the epigastrium. When the stomach rejects fluids, frequently repeated enemas of cold water are very useful. They are promptly absorbed and give great comfort to the patient (*C. B. White*). Lumbar pain and threatened suppression call for the use of turpentine stupes or sinapisms to the loins, digitalis, and mild diaphoretics. "Jaborandi is not a specific in yellow fever, but may serve certain useful indications as a sudorific and sialagogue" (*Girall*). Insomnia and nervous agitation are best relieved by bromide of potash, stimulants, and sponging of the body under the bed clothes with cistern water, to which whiskey or alcohol may be added with advantage. Chloral hydrate, or very small doses of opium are sometimes useful.

"Never try to break a return of the fever by sweating or foot-baths, but use cold sponging. If necessary move the bowels by enema. Vomiting should be met with epispastics, blisters, ice, and small doses of opium in combination with cherry-laurel water and sodæ bicarb.; hemorrhage with ergot" (*Bemiss*).

The beneficial effect of ergot in relieving hemorrhage is sometimes well marked, but administration by the stomach cannot, usually, be relied upon, as it is apt to be rejected, or at least to remain in the stomach without being absorbed. The hypodermic injection of ergotine is, therefore, more likely to succeed. In cases in which a malarial complication is suspected, or during an epidemic in a malarial neighborhood, it will be necessary to administer two or three full doses of quinine during the first twenty-four hours of sickness and to continue this remedy in smaller doses if the stomach tolerates it. During convalescence the sulphate of quinine, the citrate of iron and quinine, or the standard preparations of iron, will often be needed for their tonic effect.

Dr. Pardiñas, director of the Military Hospitals in Havana, who has had great experience in the treatment of yellow fever, gives full doses of tr. ferri chlor. throughout the disease and claims better results from this treatment than from any other he has ever tried. Whether it is better than no medicine the writer is not prepared to decide; but in view of the value of this remedy in erysipelas, diphtheria, and other malignant diseases, and of the statement of Dr. *Pardiñas* that the large doses given do not add to the irritability of the stomach, this plan of treatment seems worthy of further trial. For the relief of vomiting Dr. *Motta-Maia*, a Brazilian physician, recommends tr. nux. vom., or picric acid. The following is also recommended: Pot. iodid., grs. ij.; Liq. pot. arsenitis, gtt. ij., every two or three hours, commencing the second day (*Fowler*).

"It is the practice of some physicians to attempt the reduction of fever by large injections of cold water, which are said to prove very efficient antipyretics. Aconite, veratrum, tartate of antimony, and ipecac are frequently exhibited. A cautious use of one or the other of the first two on this list may prove beneficial, but their injudicious or careless exhibition may do irreparable harm. *We have seen digitalis produce unquestionably good effect in mitigating fever, and have often administered it in doses of thirty to sixty drops of the tincture every third or fourth hour.* It is best to give it in solutions of acetate of ammonia or potash" (*Bemiss*).

"If the patient's tongue does not clear off and nourishment begin to be craved by the close of the third twenty-four hours after the attack, *I have found it necessary to be with my patient between midnight and morning of the third night*, for if they sink at all that is the time that the sinking period will come on. The patient must be closely watched, and if the pulse has a downward tendency, *and you hear the patient sigh*, you may know that the heart's action is flagging and stimulants are required to tide them over these sinking hours" (*Walkly*).

"When one has to deal with a young man, robust, often intemperate, recently rrived from Europe, etc., . . . we will confess resolutely that, for us, the indiation appears formal in this case. It is necessary to draw blood. . . . In presence hen of a case of yellow fever of average intensity, and at the very moment of he invasion, that is to say, in the first three to six hours of the attack, I would pen largely a vein of the arm or the temporal artery and would allow a copious lood letting" (*Bérenger-Feraud*).

CROUP AND DIPHTHERIA.

BY

FRANCIS P. KINNICUTT, M.D.,

OF NEW YORK,

ATTENDING PHYSICIAN TO ST. LUKE'S HOSPITAL AND TO THE OUT-PATIENT DEPARTMENT, NEW YORK
HOSPITAL.

CROUP AND DIPHTHERIA.

BIBLIOGRAPHY.

BERTH: Zur Kenntniss d. Wunddiphtherie. Centralbl., 1873, 8, 9; Die diphth.
esse. *Ibid.*, 1873, 19.—SANDE: Ueb. gewisse ätiologische Beziehungen der
theritis zu anderen Krankheiten. Berl. klin. Woch., 1873, X., p. 315.—
SFIELD, W. H.: Connection of Diphtheria with Local Unsanitary Conditions.
Med. Journ., 1873, II., p. 655.—LETZERICH, L.: Die Entwickelung des Diph-
epilzes. Virch. Arch., 1873, pp. 303, 566.—ERHARDT, J. G.: Tracheotomy in
branous Croup and Diphtheria. Am. Jour. Med. Sci., Phil., 1873, p. 421.—
CHMEYER, E.: Zur Localbehandlung d. Diphth. Berl. klin. Woch., 1874, p.
-V. HÜTTENBRENNER: Ueb. den Catheterismus der Larynx bei der crou-
oder diphth. Erkrankung derselben. Jahrb. f. Kdrhlkde., 1874, VIII.—
EL: Im Journ. f. Kinderkr., 1874, p. 18.—SENATOR, H.: Ueb. Cynanche con-
sa. Volkm. Samml. klin. Vortr., Leipz., 1874, No. 78.—ORTH: Ueb. die Form
athogenen Bacterien. Virch. Arch., 1874, LIX., p. 532.—PANUM, P. L.: Das
de Gift der Bacterien, die putride Infection oder Intoxication u. die Septicä-
Ibid., 1874, LX., p. 301.—BILLROTH, T.: Untersuchungen über die Vegetations-
en von Coccobacteria septica. Berlin, 1874.—HILLER, A.: Der Antheil der
rien am Fäulnissprocess. Centralbl. f. d. m. W., 1874, pp. 833-849.—Report
isinfection and Disinfectants. Ann. Report of N. Y. City Board of Health,
p. 455.—LETZERICH, L.: Ueb. Abstossung einer diph. Tonsille. Berl. klin.
h., 1874, p. 339.—Microchemische Reaction des Diphtheriepilzes. Berl. klin.
h., 1874, p. 67.—VITANTONIO, G.: Il crup e la difterite. La Nuovo Ligur.
, 1874, p. 44.—CORMACK, SIR J. R.: On Diphtheritic Paralysis. Brit. Med.
n., 1874, p. 487.—BENNETT, E. D.: Cases Illustrating the Pathology of Diph-
a and Croup. Med. Press and Circ., 1874, p. 204.—SCHOTTIN, E.: Die Behand-
der Diphtheritis mit neutraler schwefeligsäurer Magnesia. Arch. d. Heilk.,
XV., p. 343.—BOUCHUT: Des infarctus pulmonaires et des abscès métasta-
s dans la diphthérite et dans le croup. Gaz. Hôp., 1874, p. 497.—MAUSORD,
Sur l'histoire de la diphthérie pharyngienne et sur les paralysies consécutives.
Paris, 1874.—LETZERICH, L.: Die locale und allgemeine Diphtherie. Virch.
., 1874, LXI., p. 457.—TRIDEAU, M. H.: Traitement de l'angine couenneuse
es balsamiques. Paris, 1874.—FONTHEIM: Die Salicylsäure gegen Diphtheri-
Memorabilien, 1874, p. 551.—JAMIESON, G.: On the Hypothesis of the
l Origin of the Contagion of Diphtheria. Austral. Med. Journ., 1874, p. 332.
EIN, A.: Zur Therapie der diphth. Rachenaffectionen. Memorabilien, 1874,
5.—NELSON, A. W.: Forty Cases of Diphtheria Treated by Local Application
lphate of Iron. N. Y. Med. Jour., 1874, p. 54.—GREENFIELD: On Diphtheria.
ent's Journ. and Hosp. Gaz., London, 1874, p. 149.—BURDON-SANDERSON,
ccurrence of Organic Forms in Connection with Contagious and Infective
ase. Brit. Med. Journ., 1875, pp. 199, 403, 435.—JENNER, SIR W.: On
p and the Diseases which Resemble it. Lancet, 1875, Vol. I., pp. 1, 75.—
SON, G.: On Certain Points Relating to Etiology, Pathology, and Treatment
iphtheria. Lancet, 1875, Vol. I., pp. 8, 80; On Croup and Diphtheria. *Ibid.*,
16, 252, 354, 423, 662, 776.—Croup and Diphtheria. Lancet, 1875, Vol. II., pp.

171, 285, 321, 353, 389, 442, 456, 529, 563, 615, 705, 845, 881. In the Lower Animals, *Ibid.*, pp. 592, 662.—**Eberth**, C. J.: Untersuch. über Bacterien. Virch. Arch., 1875, LXII., p. 504.—**Henoch**: In Ann. d. Charité, Berlin, 1875, p. 588.—**Hiller**, A.: Experiment. Beitr. zur Lehre v. d. Fäulniss. Arch. klin. Chirur., 1875, XVIII., 669.—**Monti**: Ueb. Croup im Kindesalter. Wien. Klinik, 1875, 1 u. 2 Heft.—**Satterthwaite**, **Th.** E.: Bacteria, Their Nature and Relation to Disease. Med. Rec., 1875, pp. 833, 849.—**Satterthwaite**, **Th.** E. and **Curtis**, E.: *Ibid.*, 1875, Dec. 18th and 25th.—**Fleck**, H.: Vergleichende Versuche zur Feststellung d. Werthes der Salicylsäure als Disinfectionsmittel, etc. München, 1875.—**Field**, A. G.: Cellars and Diphtheria, etc. Med. Rec., 1875, p. 820.—**Bouchut**: Signes ophthalmoscopiques dans les paralysies diphthéritiques. Gaz. Hôp., 1875, pp. 667, 673.—**Cormack**, **Sir** J. R.: Diphth. Paralysis, its Natural Course, Pathology, etc. Brit. Med. Journ., 1875, pp. 133, 571, 670.—**Castle**, F. D.: A Case of Paresis of the Ciliary Muscle Following Diphtheria. Phil. Med. Times, 1874–75, p. 230.—**Pooley**, J. H.: Tracheotomy in Diphtheria. West Va. Med. Student, 1875, p. 37.—**Letzerich**, L.: Exper. Untersuch. u. Beobachtungen über die Wirkung d. Salicylsäure bei d. Diphtherie. Virch. Arch., 1875, LXIV., p. 102.—**Smith**, J. L.: Diphtheria, Causes and Nature of, with a Review of the Bacterian Theory. Sanitarian, N. Y., 1874–75, p. 509.—**Bizzozero**, G.: Contribuzione all' anatomia patologica della difterite. L'Osservatore, 1875, p. 497.—**McReady**, R. and R. J.: Two Cases of Recovery from Diphth. Croup, One With, and One Without Tracheotomy. Amer. Journ. Med. Sci., 1875, p. 417.—**Hüter**: Ueb. d. experiment. Erzeugung der Diphtheritis. D. Ztschr. f. Chir., 1875, p. 624.—**Rignault**, G.: Du traitement local des affections couenneuses par l'acide lactique. Rev. Thérap., 1875, p. 567. —**Lusk**, W. J. H : Notes on a Case of Diph. Followed by Paraplegia, Recovery. Med. Times and Gaz., 1875, p. 361.—**Bartholow**, R.: Diph. Paralysis. Clinic Cinc., 1875, p. 49.—**Lyle**, W. V.: Notes on a Recent Outbreak of Diph. Lancet, 1875, p. 460.—**Day**, J.: Is Diphtheria Primarily a Blood Disease? Med. Times and Gaz., 1875, p. 337.—**Jacobi**, A.: Contributions to the Pathology and Therapeutics of of Diph. Journ. of Obstet., 1875, Feb.—**Letzerich**, L.: Fall v. Diphtherie der Impfwunden, allgemeine Diphtherie, Tod, etc. Virch. Arch., 1875, LXIII., p. 178.—Diphtheria in N. Y. Med. Rec., 1875, p. 425.—**Henoch**: Klin. Mittheilungen über Diphtherie. Berl. klin. Woch., 1875, p. 217.—**Bastian**, H. C.: Microscopic Germ Theory of Disease, etc. Monthly Micros. Journ., 1877, pp. 65, 129; Lancet, 1875, p. 501; Brit. Med. Journ., 1875, p. 469.—**Birch-Hirschfeld**, V.: Neuere pathologisch-anatomische Untersuch. über Vorkommen und Bedeutung niederer Pilzformen bei Infections-Krankheiten. Schmidt's Jahrb., 1875, p. 169.—**Stimson**, L. A.: Bacteria and their Influence on the Origin and Development of Septic Complications of Wounds. N. Y. Med. Journ., 1875, p. 113.—**Klebs**, E.: Beitr. zur Kenntniss der pathologen Schizomyceten. Arch. f. exp. Path. u. Pharm., 1875, IV., p. 305.—**Davaine**: Sur la découverte des bacteridies. Bull. de l'Acad. de Méd., 1875, p. 581.—**Moxon**, W., and **Goodhart**, J. F.: Observations on the Presence of Bacteria in the Blood and Inflammatory Products of Septic Fever, etc. Guy's Hosp. Reports, 1875, p. 229.—**Hiller**, A.: Ueb. diagnostische Mittel u. Methoden zur Erkennung der Bacterien. Virch. Arch., 1875, LXII., p. 361.—**Billroth**, **Th.**, und **Ehrlich**, F.: Untersuch. über Coccobacteria septica. Arch. f. klin. Chir., 1876, XX., p. 403.—**Benson**, S.: Chlorine Water in Diphtheria. Canada Lancet, 1876, p. 169.—**Oertel**: Epidem. Diphtherie. Im Handb. v. Ziemssen, 1874 u. 1876.—**Welch**, W. M.: Are Croup and Diphtheria Identical? Phil. Med. Times, 1876, p. 193.—Croup and Diphtheria. Brit. and For. Med. Chir. Rev., 1876, p. 102.—**Cohen**, J. S.: Diphtheria. Med. Rec., 1876, Feb. 12th and 19th.—**Moir**, J.: Diphtheria. Brit. Med. Journ., 1876, Vol. I., pp. 345, 618.—**Semple**: Relation between Croup and Diphtheria. *Ibid.*, Vol. I., p. 431.—Diphtheria, Treatment of. Lancet, 1876, Vol. I., p. 800.—**Billington**, C. E.: Diphtheria and its Treatment. Trans. N. Y. Acad. Med., 1876.—**Bizzozero**: Beitr. zur pathol. Anat. der Diphtheritis. Oester. med. Jahrb., 1876, Heft 2.—**Betz**: Die

Eisbehandlung beim Croup. Memorabilien, 1876, No. 10.—SCHÜLLER: Ueber d. Stellung der Tracheotomie zum Croup. D. med. Woch., 1876, Nos. 36 und 37.—TEUHOLT: Diphtheritis Treated by Salicylic Acid and Lime-Water. Practitioner, 1877, Vol. XVIII., p. 298.—Croup, Treatment of, by Eucalyptus. Brit. Med. Journ., 1877, Vol. I., p. 393.—JACOBI, A.: Diphtherie. Handb. der Kinderkrankh., Tübingen, 1877, II. Band.—SANNÉ, A.: Traité de la diphtherie. Paris, 1877.—KRÖNLEIN: Diphtheritis u. Tracheotomie. Langenbeck's Arch. f. Chir., 1877, Vol. XXI.—WEIGERT: Ueb. Croup u. Diphtheritis. Virch. Arch., 1877, p. 461.—SEITZ, F.: Diphtherie und Croup. Berlin, 1877.—VIRCHOW, R.: Diphtherie und Croup. Berl. Charité-Annalen, 1877, Vol. II., p. 710.—LAMB: Cases of Diphtheria and Croup. Guy's Hosp. Reports, 1877, Vol. XXII., p. 345.—KRIEGER: Aetiologische Studien. Strassburg, 1877.—Croup, Tracheotomy in. Brit. Med. Jour., 1877, Vol. II., p. 633.—Diphtheria, Influences Affecting Propagation of. *Ibid.*, p. 302.—RAUCHFUSS: Die Krankh. des Kehlkopfes, etc. Handb. d. Kinderkrankh., 1878, Vol. III.—KOCH, R.: Untersuch. über d. Aetiologie d. Wundinfections-Krankheiten, 1878.—Diphtheria, Contagiousness of. Brit. Med. Journ., 1878, Vol. I., p. 201.—Diphtheria, Specific Origin of. *Ibid.*, Vol. II., p. 367.—Diphtheria. Lancet, 1878, Vol. I., p. 290. Origin of. *Ibid.*, Vol. II., p. 830.—Croup. Practitioner, 1878, Vol. XX., p. 47.—DOWNES, A.: Diffusion of Diphtheria. Brit. Med. Journ., 1878, Vol. II., p. 920.—HOVELL, D. DE B.: Treatment of Diphtheria. Lancet, 1878, Vol. II., p. 914.—MCEWEN, F. A.: Internal Administration of Carbolic Acid in Diphtheria. Practitioner, 1878, pp. 428, 430.—SCHECH: Doppelseitige complete Recurrenslähmung nach Diphtherie. D. Arch. f. klin. Med., 1878, pp. 166–171.—MACKENZIE, M.: Diphtheria, etc. London, 1878, 8vo.—CARPENTER, J. T.: On the Identity of Hospital Gangrene with Diphtheria. Trans. Amer. Med. Ass., 1878; Vol. XXIX., pp. 237–245.—Reports of Three Outbreaks of Diphtheria in Michigan. Rep. Bd. Health, Mich., 1878, VI., pp. 75–89.—KLEBS: Natronbenzoat u. Diphth. Prager med. Woch., IV. Jahrg., 1878, Nos. 3 u. 5.—LETZERICH: Diphth. u. Natronbenzoat. Berl. klin. Woch., 1879, p. 93.—HOFFMAN, L.: Ueb. d. therap. Wirkung des Natron benzoicum. Berl. klin. Woch., 1879, p. 225.—SIMON, J.: Essay on Contagion. Lancet, 1879, pp. 926, 973.—STEINER, J., und V. ZIEMSSEN: Laryngitis crouposa et diphtheritica. Handb. d. spec. Path. u. Therap., 1879, Vol. IV., 1 Hälfte.—CARPENTER, A.: A Possible Predisposing Cause of Diphtheria. Brit. Med. Journ., 1879, Vol. I., p. 8.—DAVIS, C. H. S.: Diphtheria and its Treatment. Louisville Med. Journ., 1879, pp. 97–134.—FIELD, J.: An Inquiry into the Cause of Diphtheria. *Ibid.*, pp. 15–21.—GOLDSCHMIDT, S.: Ueb. das Zusammentreffen v. Scharlach u. Diphtheritis. Centr. Ztg. f. Kinderk., 1879, pp. 132–135.—ROBINSON, W. L.: Use of Oxygen Gas in Treatment of Diphth. Tr. Med. Soc. Virginia, 1879, II., 454–456.—CAMERON, H. C.: Proneness of Certain Families to Contract Diphtheria. Brit. Med. Journ., 1879, I., 270.—CORBY: Are Croup and Diphtheria the Same Disease? Dublin J. M. Sc., 1879, 3 s., LXVII., 172–175.—Diphtheria and Milk. Med. Times and Gaz., 1879, I., 66.—EIGENBRODT: Report of the Med. Hist. of the Attack of Diphth. in the Grand-Ducal Family of Hesse-Darmstadt. Brit. Med. Journ., 1879, I., 6, 112, 147, 187, 268.—FLEMING, G.: "Garget" and Diphtheria. Lancet, 1879, I., 137.—Garget and Diphtheria. San. Record, Lond., 1879, X., 38.—HICKS, J. A.: Treatment of Diphtheria. Lancet, 1879, I., 69.—HOLLAND, E.: Propagation of Diphtheria, etc. Brit. Med. Journ., 1879, I., p. 148.—FORGET, A.: Recherches expér. sur l'emploi du perchlorure de fer dans la diphthérie. Paris, 1879, 32 pp. SEMPLE, R. H.: Diphtheria, etc. Lond., 1879.—BROWNE, R. F. C.: Kali permanganicum Administered Hypodermically in Malignant Diphtheria. U. S. Med. Invest., Chicago, 1879, n. s., IX., pp. 206–208.—GARNETT, J. N.: Treatment of Diphtheria by Carbolic Acid and Iodoform. Amer. J. M. Sc., Phil., 1879, n. s., p. 461.—HOCK, J.: Sind Croup u. Diphtheritis identische Krankheitsprocesse? Wien. med. Blatt, 1879, II., 181.—VIRCHOW, R.: Angina, Diphtheria u. Croup. Charité-Anna., Berl., 1879, IV., pp. 751–759.—Report of Scientific Committee

Appointed by the Roy. Med. and Cirurg. Soc. to investigate the Relation between Membranous Croup and Diphtheria. Lond., 1879.—Alloway, T. J.: Two Successful Cases of Tracheotomy in Laryngeal Diphth. Canada Med. Rec., 1879, VII., p. 141.—Diphtheria, its Dissemination by Milk, Milk-Kine, and Poultry. Practitioner, 1879, pp. 306–315.—Downes, A.: Diphtheria Diffusion. Lancet, 1879, I., 515.—Diphtheria and Milk Supply. Brit. Med. Journ., 1879, I., 148.—Lownds, T. M.: On Treatment of Diphtheria. Lancet, 1879, I., 403.—Stuart, J. A. E.: Sulphur as a Topical Application in Diphtheria. Practitioner, 1879, XXII., 248; XXIII., 272.—Blake, E. F.: Is Diphtheria Preventable, etc.? London, 1879, 24 pp., 6 pl.—Browning, B.: Water as an Agent in the Spread of Diphtheria. Med. Times and Gaz., 1879, I., 520.—Davies, H.: Diphtheria and Polluted Water. San. Rec., Lond., 1879, X., p. 269.—Diphtheria and Bad Drainage. *Ibid.*, p. 275. —Discussion on Croup and Diphtheria. Med. Times and Gaz., 1879, I., pp. 437, 488, 539, 577; Brit. Med. Journ., 1879, I., pp. 588, 664, 776–779; Lancet, 1879, I., pp. 556, 593, 628, 701, 735.—Nicati, W.: Sur diverses épizooties de diphthérie des oiseaux de basse-cour observées à Marseille, etc. Compt. rend. Acad. d. Sc. Par., 1879, pp. 297–299; Marseille méd., 1879, pp. 104–107.—Sur une cause possible de propagation de la diphthérie, etc. Rev. d'hyg., 1879, pp. 237–243.—Closson: Du benzoate de soude dans la diphthérie. Ann. Soc. méd.-chir. de Liége, 1879, pp. 221–228.—Dickinson, W. H.: Croup and Diphtheria. Med. Times and Gaz., 1879, I.—Report of Committee Appointed to Investigate the Relations of Membranous Croup and Diphtheria. Medico-Chirurg. Trans., 1879, LXII., pp. 1–168; Proc. Roy. Medico-Chirurg. Soc., 1879, VIII., pp. 348–351.—Bouffé, F.: Recherches cliniques sur la diphthérie, et de son traitément en particulier. Paris, 1879.—Recent Reports on Certain Outbreaks of Diphtheria and Enteric Fever. Med. Press and Circ., London, 1879, pp. 248, 250.—Triboulet: 108 Autopsies de Diphthérie a l'hôpital Sanité-Eugénie. Progrès méd., Paris, 1879, VII., pp. 744–752.—Johnson, G.: Membranous Croup and Diphtheria. British Med. Journ., 1879, I., 848.—Williams, P. C.: Diphtheria and its Relation to Membranous Croup. Maryland Med. Journ., 1879–80, V., pp. 77–86.—Schüssler: Heilung d. Diphtheritis auf biochemischen Wege. Oldenberg, 1879.—Packard, J. H.: Diphtheria apparently Caused by Exposure to the Poison of Scarlatina. Amer. Journ. of Obstet., 1879, p. 641.—Semple, R. H. : Diphtheria and Croup. Med. Press and Circ., London, 1879, p. 475.—Wells, E. F.: Diphtheria: an Analysis of over 300 Cases. Cincin. Lancet and Clinic, 1879, III., pp. 41–47.—Widerhofer: Ueber d. Wirksamkeit des benzoësauren Natron bei Diphtheritis. Wien. med. Bl., 1879, II., 575, 610, 642.—Map showing a Portion of Winstead, Ct., Showing Location of Fatal Cases during the Diphtheria Epidemics of the Fall of 1878. Proc. Ct. Med. Soc., Hartford, 1879, LXXXVIII., 55.—Mossler, F.: Ueber Inhalation des Oleum Eucalypti bei Rachendiphtherie. Berl. klin. Wochenschr., 1879, pp. 301–304; Allg. med. Centr.-Ztg., Berl., 1879.—Talamon, C.: Diphthérie œsophagienne dans un cas de diphthérie infectieuse, etc. Progrès méd., Paris, 1879, VII., 204.—Gilman, J. H.: Diphtheria. N. Y. Med. Rec., 1879, p. 269.—Burrall, F. A.: Diphtheria and Benzoate of Soda. *Ibid.*, XVI., p. 572.—Haddon, J.: Diffusion of Diphtheria. British Med. Journ., 1879, II., 813. —Kien, A.: De la Diphthérie, en particulier de son traitement par le benzoate de soude. Gaz. med. de Strasb., 1879, VIII., pp. 149–153.—Smith, L.: Pathol. Differences of Diphtheria and Membranous Croup. Med. Rec., 1879, XVI., p. 594.—Smith, A. H.: Supplementary Rectal Alimentation. Arch. of Med., N. Y., 1879, Vol. I., p. 113.—Sainclair, P.: Contribution à l'étude de la pathogénie des paralysies diphthéritiques. Lyon, 1879, 86 pp.—Jakobi, A. I.: Osanitarnich mierach po povodu epidemii difterita v. Achstrskom niezlie, char. kovskoi gubernii [Precautions against diphtheria]. Sborn. sochni. po sudebrioi med. St. Petersb., 1879, III., pp. 68-74.—Payne, R. L.: Limitation and Prevention of Diphtheria. N. Carolina Bd. of Health, 1879, 10 pp.—Scarpari, S.: Contribuzione allo studio delle paralisi difteriche. Ann. univ. di med. e chir., Milano, 1879.

pp. 369–392.—Lucas, J. C.: Croup and Diphtheria. Indian M. Gazette, Calcutta, 1879, XIV., pp. 252–255.—Ogle, W.: Outbreaks of Diphtheria in Rural Districts. St. Geo. Hosp. Rep., 1879, IX., pp. 701-726.—Wells: Diphtheria with Long Period of Incubation. Boston M. and S. Journ., 1880, p. 346.—Meyer, J.: Om Behandlingen af Difteritis i Svœlget [On treatment of diphtheria]. Hosp. Tid. Kjöbenh., 1880, VII., 207-209.—Discussion on Relation of Croup and Diphtheria. Brit. Med. Journ., 1880, I., pp. 301–303.—Billington, C. E.: 40 Attested Cases of Diphth.: Diagnosis and Treatment. Med. Rec., 1880, XVII., pp. 333–342. Treatment of Diphtheria. *Ibid.*, p. 383.—Jochheim, Ph.: Diphtheritis u. Ozon. Neue erfolgreiche Behandlung der Diphtheritis. Heidelb., 1880.—Schuster, C.: Beitr. zur Behandlung der Diphtherie. D. med. Woch., 1880, VI., pp. 30–32.—Diphtheria. Bd. of Health Circular, Boston, Jan., 1880, 2 pp.—Diphtheria in Russia. Brit. Med. Journ., 1880, I, 217.—Griffiths, A. L.: How Diphtheria is Spread. Lancet, 1880, I., 273.—Merlini, P.: Epidemia difterica; frequenza ed importanza dell' albuminuria; croup e difteria. Arch. Clin. Ital., Roma, 1880, X., 1, 13.—Greenfield, W. S.: Recent Investigations into Pathol. of Infective and Contagious Diseases. Lancet, 1880, pp. 83, 355, 517, 631.—Moir, J.: Croup: its Nature and Treatment. Edinb. Med. Journ., 1879–80, XXV., pp. 899–909.—Bouchut: Traitement des paralysies diphthéritiques. Gaz. d. Hôp., 1880, LIII., p. 370.—Landowski, P.: L'angine diphthérique et la croup au point de vue clinique. Journ. d. Therap., 1880, VII., pp. 253–260.—March, C. J.: Is Diphtheria Capable of being transported by Fomites? Repts. of cases, etc. St. Louis M. and S. Jour., 1880, pp. 406–408.—Buchanan, G.: Tracheotomy in Diphth. and Croup.—Brit. Med. Journ., 1880, p. 554.

The design of this article is simply to present a concise résumé of the literature of croup and diphtheria, since the publication of the first edition of *v. Ziemssen's* Cyclopædia. The limited space allotted to the article has necessarily restricted the writer to a brief and sometimes imperfect analysis of the experience and observations recorded in this special field of pathology. It is hoped, however, that the article will fairly represent the most trustworthy and valuable additions which have been made during the past ten years to our knowledge of the causes, pathology, and treatment of these important diseases.

THE GERM THEORY OF DIPHTHERIA.

The theory of a specific living organism constituting the contagium of diphtheria originated in the simultaneous discovery by *Oertel, Hüter,* and *Buhl,* in 1868, of minute living organisms in the diphtheritic membrane, in the subjacent diseased tissues, and in the blood. The vegetable organisms observed belong to a group known to botanists as schizomycetes, which, after *Cohn,* may be roughly classified as follows: spheroidal bacteria (micrococcus); rod-shaped bacteria (bacterium termo); long rod bacteria or bacilli; spiral or twisted bacteria, of which the type is spirillum. The forms chiefly observed in diphtheria are the micrococcus and bacterium termo. On the announcement of the discovery, a series of researches was undertaken by numerous German pathologists, which established the fact of the very constant presence of these micro-organisms, not only in the diphtheritic membrane, but also in the blood and various tissues and organs of those dying of the disease (*Von Recklinghausen,* *Nassiloff, Waldeyer, Heiberg, Eberth, Tommasi, Klebs,* and others). The inoculability of the disease was proven in the earlier experiments, by the successful infection of animals by means of fresh diphtheritic membrane (*Von Trendelenberg* and *Nassiloff* in the trachea and on the cornea, *Eberth* on the cornea, *Hüter* and *Tommasi* in the larynx and pharynx, *Oertel, Tommasi, Hüter, Letzerich* in the muscles). As a result of these pathological experiments, a causal relation to the diphtheritic process was asserted for the bacterial growths. Further investigations were undertaken to solve the disputed question. *Eberth's* successful removal of the infectious properties of a virulent diphtheritic infusion by means of thorough filtration, an experiment subsequently repeated by other observers with similar results, apparently proved at least the *particulate* nature of the diphtheritic poison. *Klebs* introduced the ingenious method of fractional cultivation into his experiments. He successfully inoculated animals with the bacteria of diphtheria obtained in this manner, and was able to recognize the same in their blood and tissues after death. *Rindfleisch,* in the last edition of his Pathologische Gewebelehre (1878), having described the successful inoculation of the cornea of rabbits, after *Eberth's* method, with the "micrococcus diphtheriticus," concludes as follows: "We could hardly desire a more binding and convincing proof of the mode of penetration and general diffusion of the diphtheritic germ (pilz) into the body."

Letzerich differs from other German observers in regarding a true *fungus,* of the hyphomycetes family, as the specific contagium of diphtheria.

The English investigators, *Ewart* and *Simpson,* claim that the diphtheritic poison consists of minute spores, scarcely larger than the smallest forms of micrococci, but capable of germinating into long, very fine bacilli on being deposited in a suitable medium, such as is provided by the surface of the tonsils, the soft palate or pharynx, when deprived from any cause of their superficial epithelium.

The morphological similarity of the micro-organisms of diphtheria and those which have been found to be present in the various wound-infection diseases (pyæmia, septicæmia, hospital gangrene) is generally admitted; at the same time it is justly claimed that this resemblance may be due to our still imperfect methods of observation.

The principal arguments of the opponents of the bacterial origin of infectious and contagious diseases may be summarized as follows: *Billroth* has attempted to prove the identity of the various forms of bacteria, and is disposed to believe that living blood is destructive to their life.

Hiller's conclusions from his numerous pathological experiments are to the effect that it is impossible to identify multiplying bacteria with the septic process; they may be imbibed with the septic poison and become carriers of it, but they are incapable of independent action.

Charlton Bastian's advocacy of the physico-chemical theory of contagion is well known. The evidence which he adduces to disprove the existence of any causal relation between low forms of organism and the various septic and contagious diseases is as follows: (a.) Bacteria may be

Professors *Klebs* and *Tommasi*, in recent researches conducted at Rome, have succeeded in isolating from malarious soils and their atmosphere specific microphytic forms (bacillus malariæ), and from their inoculation, after successive cultivations, have been able to produce true ague in animals. Finally, in a communication to the French Academy of Sciences during the past winter, M. *Pasteur* describes a specific microscopic organism, first discovered by M. *Toussaint*, Professor in the Veterinary School of Toulouse, as present in the "cholera of fowls," and asserts that he is able to produce the disease by their inoculation, after artificial cultivation.

THE GENERATION AND MEANS OF TRANSMISSION OF THE DIPHTHERITIC POISON.

Within the past two years, the investigations of Mr. *Power*, of London, in regard to the possible dissemination of diphtheria by milk, have excited much merited attention.

Mr. *Power's* investigations were undertaken in behalf of the Local Government Board, in regard to an outbreak of diphtheria in a North London district. Within a circumscribed area, which could be inclosed by a circle of a mile and a half in diameter, there occurred, between March 2d and June 15th, 264 cases of diphtheria in 118 households, and of these 78, numbering 186 cases, were attacked during the four weeks ending May 25th. It was thus evident that the etiological factor was also circumscribed in area, and operative during a very limited period. A most rigorous investigation of the hygienic status of the district, of the influence of communication between the sick and well, failed to afford a more satisfactory clue than that the sewage of a portion of the affected area had been temporarily disturbed, five weeks previous to the outbreak of the disease.

Early in the outbreak, Dr. *Morton*, in whose practice many of the cases occurred, was impressed with the fact that a very large proportion of the persons infected used the milk supplied from the same source. The investigation, prompted by this suggestion, established the following remarkable facts: Of 430 households in the affected district, supplied by milk from the same source, 64 were invaded, or 148 per 1000; of 1870 households supplied by milk from a different source, 22 were invaded, or 11 per 1000, a difference of 13 to 1. It should be mentioned that a small portion of the milk supplied to the 1870 households was also obtained from the first-mentioned dairy. It is further shown that not only did the consumers of this dairy "suffer relatively and actually in excess of other people, but that these persons bore almost the whole brunt of the outburst of the throat illness, which first attracted attention to the district, and which had been locally referred to sewer causation." Mr. *Power* believes, as the result of his investigations, that the connection between the distribution of diphtheria in the above outbreak, and the distribution of a particular milk, is proven, and he is disposed to believe that there was an infectiveness of the milk *per se*, rather than one due to the transference to it of any conditions external to the cow. He, as well as Mr. *Smee*, suggests the importance of a study of the disease of the cow known as garget, in this connection.*

During the year 1879, several outbreaks of diphtheria, in different parts of England, were very distinctly traced to the milk supply of the infected districts; the source of the milk-pollution or milk-infection, however, was not clearly demonstrated.

Simultaneously with the investigations in regard to the possible relation of an undetermined disease of the cow to diphtheria in man, Dr. *Nicati*, of Marseilles, suggested a similar relationship between the diphtheria of fowls and the diphtheria of the human being. A careful study of the disease of the poultry-yard convinced him as to its true diphtheritic character. He successfully inoculated various animals with the false membrane, and also showed that the outbreak of the malady among the fowls coincided with an increase of diphtheria among the inhabitants of the city. In conclusion he observes, "this coincidence between diphtheria of poultry and that of the human species, the analogy in the clinical characters of the two diseases, and the inoculability of the diseases by means of the false membranes, render their identity more and more probable," and he advises, in consequence, "the rigorous inspection of all poultry at the time of their being brought into the city."

The medical blue books of the English government have contained almost yearly, since 1866, reports of investigations of various outbreaks of diphtheria throughout the kingdom. They show the coincidence of many of them with the existence of soil and house dampness, particularly when associated with conditions favoring animal and vegetable decay and with defective sewage. An explanation of the *spread* of the disease, in many of the outbreaks, is easily found in personal infection and through infected bedding and clothing; but a very careful study of the numerous reports does not justify us in claiming for the unfavorable hygienic conditions present, a more than predisposing influence in the *causation* of diphtheria.

In an outbreak investigated by Dr. *Thorne Thorne*, the important fact was shown that apparently simple non-infectious sore throat "was capable, under the circumstances of this outbreak, of leading to more severe and infectious sore throats, and that these in turn, by direct infection, gave rise to well-marked and even fatal cases of diphtheria." In other words, that "the property of infectiveness was capable of progressive development."

Sanne, in his elaborate treatise on diphtheria, claims that evidence is lacking of a *de novo* origin of the disease. He believes that contagion is the most probable mode of propagation, using the term in the sense of transmissibility, mediate or immediate. He asserts that reports of physicians from various parts of France show that diphtheria first appeared in their departments with the establishment of railways.

Seitz, while believing in the contagiousness of diphtheria, mediate and immediate, at the same time is disposed to admit the possibility of its spontaneous origin. He grants that the conditions of such an origin are wholly unknown, that they are not those of weather, soil, or climate. Professors *Jacobi* and *Bystrow*, of Russia, in discussions before the Medical Society during the past winter, in regard to the present widely-spread and very fatal epidemic of diphtheria in that country, asserted that the disease spreads, not only by contact of the healthy with the sick, but also by means of the infected clothing of the latter, and earnestly urged the necessity of its destruction or thorough disinfection. The very general admission, at the present time, by authorities in various parts of the world, of this mode of transmission of the diphtheritic poison may justly be considered as a possible explanation of many hitherto inexplicable outbreaks of the disease.

---

* A mammitis, one form of which has been shown to be infectious.

The fact of the long preservation of the virulence of the contagion of certain other infective and contagious diseases lends itself as evidence in favor of the possession of a similar power by the diphtheritic poison.

In conclusion, it may be claimed that, from the evidence accessible at the present date, *a de novo origin of the contagium of diphtheria is not proven, as a consequence either of atmospheric or telluric influences, of soil or water pollution, or of defective drainage;* on the other hand, *accumulating evidence seems to point to the specific nature of the diphtheritic poison, and to its transmission through contagion, mediate or immediate.*

THE RELATIONS OF CROUP AND DIPHTHERIA.

A brief résumé of modern views in regard to the relations of membranous croup and diphtheria is necessary for a comprehension of the present phase of this much vexed question.

It will be convenient first to consider the anatomical distinctions between croup and diphtheria. English pathologists, in seeking for a distinction of this kind, assumed that the croupous membrane was a simple fibrinous exudation, and with this the diphtheritic membrane was compared. The peculiarities of the latter, which *Harley*, *Murchison*, *Hillier*, and others pointed out, more modern investigation has shown to be shared by croupous exudation. In other words, English observers have failed to establish an anatomical basis for the distinction of croupous from diphtheritic products.

In Germany, the original anatomical definition given to croup by *Rokitansky* was that of "a fibrinous exudation effused in a liquid form, and coagulating on the surface of the mucous membrane, this being unaltered or nearly so." On the other hand, diphtheria was described as "a necrotic process, consisting in infiltration of the mucous membrane, accompanied by exudation and followed by sloughing." Through *Virchow's* teachings mainly, the meaning of the terms croupous and diphtheritic was widely extended. The former term was applied to all inflammations of mucous membranes accompanied by fibrinous exudation upon the surface; while the latter was used to designate processes consisting in infiltration with loss of substance. *Wagner* regarded pharyngeal diphtheria and croup of the air passages as examples of diphtheritic and croupous inflammations respectively. The croupous membrane was shown by him to be composed of a thick network of delicate, very fine threads, whose interstices are occupied by numerous bodies resembling pus-corpuscles. He believes that the origin of this network is in a peculiar metamorphosis of the epithelial cells; the cells enlarge and send out prolongations, by the coalescence of which the network is formed. The subjacent mucous membrane is hyperæmic and infiltrated with cells, but chiefly in its upper layers. The diphtheritic membrane is composed of a similar network, also inclosing elements resembling pus-corpuscles, which are much less numerous, however, than in the croupous membrane. The network is produced, as in croup, by a cell-metamorphosis. The mucous membrane, the submucous, and sometimes even the connective tissue are infiltrated with newly-formed cells and nuclei. In other words, the distinction between the croupous and diphtheritic inflammation is really one of degree. *Wagner* believes that croupous as well as diphtheritic exudations may occur on the gums and throat; that more frequently, however, the latter is seen in the throat, the former in the lower part of the larynx, the trachea, and bronchi, while an intermediate form is found in the upper part of the larynx. *Wagner*, therefore, makes no anatomical distinction between the *diseases* croup and diphtheria. *Steiner*, the author of the article on croup in the first edition of v. *Ziemssen's* Cyclopædia accepts the results of *Wagner's* investigations, and asserts that the attempt to separate diphtheria and croup as two entirely distinct diseases has been unsuccessful, both from an anatomical and clinical stand-point. *Von Ziemssen* has re-written the above article in the last edition of the Cyclopædia and expresses a similar view.

Rindfleisch, in the article on angina diphtheritica, in the latest edition of his work (1878), describes the formation of pseudo-membranes upon mucous surfaces and calls them *croupous* deposits. According to him, surface exudations occur, fall off or are removed, and their place is filled by others, which gradually lose their superficial character, inasmuch as their under-surface involves the parenchyma of the mucous membrane; their separation is accompanied by loss of substance, and this one step is, according to many authors, the differentiating factor between a croupous and diphtheritic inflammation. *Rindfleisch* believes that the pseudo-membrane is the result of a separation of young elements from the surface of an irritated mucous membrane, which have undergone a peculiar form of degeneration, the exact nature of which is not yet understood. Diphtheritis is to him an etiological entity, dependent upon the emigration of certain organisms of the lowest type into the blood and tissues of the body; it is, therefore, to be considered as an infectious general disease, the local lesions being due to a *croupous* inflammation of the mucous membrane at the point of entrance and location of the organisms. According to *Rindfleisch*, "the distinction between croup and diphtheria, in the older sense of the words, is founded upon the anatomy of the parts involved."

In France, the identity of croupous and diphtheritic exudations was first asserted by *Bretonneau*. A similar view is held by French and very generally by Italian pathologists at the present time.

In America, as early as 1860, *Jacobi* also expressed his belief in their identity. He showed that the exudations might merely be adherent to the mucous membrane, without alteration of its tissue, as is usually the case when they occur in the bronchi, trachea, and on the soft palate, or that they might be imbedded in its substance, as is observed on the tonsils and the posterior pharyngeal wall, as well as occasionally in the larynx.

It will thus be seen that the investigations of pathologists fail to furnish *an anatomical distinction between membranous croup and diphtheria.*

The elaborate researches undertaken under the auspices of the Royal Medical and Chirurgical Society of London, and only recently completed, to determine the relations of membranous croup and diphtheria, constitute a most exhaustive study of the question from a clinical stand-point. The object of the inquiry, as stated by the committee appointed by the society, is to determine "whether membranous laryngitis exists independently of the diphtheritic poison, and whether, if so, there are any criteria by which it can be distinguished clinically or pathologically." The experience of the Fellows of the society, as well as of distinguished observers in all parts of the world, was sought, and a large mass of information was thus collected. A digest of the replies received and a critical summary of them was presented by the committee. In the investigation the following points were considered.

1. The known causes of membranous laryngitis and their relative frequency.
2. The conditions of occurrence.
3. The possible distinctions between classes of cases, as regards general course, symptoms, the anatomical distribution of the false membrane, etc.

The general admission was elicited that the poison of diphtheria holds the first rank among the causes of membranous laryngitis, and that there is a laryngeal form of diphtheria in which the disease is wholly limited to the larynx and trachea, the ratio being about one in thirty cases. The number of cases in which membranous laryngitis was apparently produced by mechanical or chemical irritants was found to be very small and was not thought to be available for statistical consideration.

Conditions of occurrence.—Upon these points the committee received little information. Notwithstanding that every effort was made to elicit evidence of the occurrence of membranous laryngitis from exposure to cold, no single case was furnished to the committee; nor "did the replies on any of the other points on which inquiry was made contribute anything to the knowledge of membranous laryngitis."

The possible distinction between classes of cases, etc.—Great difficulty was experienced in dealing with this portion of the subject from the absence of positive symptoms by which, provisionally, to separate classes for inquiry. The anatomical distribution of the false membranes was finally accepted as a criterion. The value of conclusions based upon such a test was justly considered as impaired by the following facts. (1) Membranous laryngitis is especially a disease of infancy and childhood, and from the impracticability of a thorough examination of the pharynx and posterior nares of children, exudations may exist in these situations unsuspected. (2) The discovery of false membrane, limited to the larynx, on post-mortem examinations, does not exclude a possible implication of the pharynx at the outset of the disease.

Suddenness of onset was shown to be rather a characteristic of non-membranous laryngitis than of different forms of the membranous variety.

Albuminuria was found to be relatively less frequent in cases of laryngeal exudation alone, but the fact was elicited that it also occurred in cases of laryngitis in which no membrane is formed. The early fatality of membranous laryngitis and the possibility that the laryngeal affection may modify its presence, was considered as rendering this criterion indecisive.

Swelling of the glands appeared to be more frequent in the faucial than in the laryngeal affection, but it was also shown to be often absent in the former.

The summary of the committee's conclusions is as follows:

"1. Membranous inflammation confined to or chiefly affecting the larynx and trachea may arise from a variety of causes.

"(a.) From the diphtheritic contagion.

"(b.) By means of foul water or foul air, or other agents, such as are commonly concerned in the generation or transmission of zymotic disease (though whether as mere carriers of contagion cannot be determined).

"(c.) As an accompaniment of measles, scarlatina, or typhoid, being associated with these diseases independently of any ascertainable exposure to the special diphtheritic infection.

"(d.) It is stated on apparently conclusive evidence, although the committee have not had an opportunity in any instance of examining the membrane in question,* that membranous inflammation of the larynx and trachea may be produced by various accidental causes of irritation, the inhalation of hot water or steam, the contact of acids, the presence of a foreign body in the larynx, and a cut throat.

"2. There is evidence in cases which have fallen under the observation of the committee, that membranous affection of the larynx and trachea has shortly followed exposure to cold, but their knowledge of the individual cases is not sufficient to exclude the possible intervention or co-existence of other causes. The majority of cases of croupal symptoms definitely traceable to cold appear to be of the nature of laryngeal catarrh.

"3. Membranous inflammation, chiefly of the larynx and trachea, to which the term 'membranous croup' would commonly be applied, may be imparted by an influence, epidemic, or of other sort, which in other persons has produced pharyngeal diphtheria.

---

* One such case was communicated to the committee subsequent to the completion of the report, and the membrane was examined.

"4. And conversely, a person suffering with the membranous affection chiefly of the air passages, such as would commonly be termed membranous croup, may communicate to another a membranous condition limited to the pharynx and tonsils, which will commonly be regarded as diphtheritic.—In conclusion, the committee suggest that the term *croup* be henceforth used wholly as a clinical definition implying laryngeal obstruction occurring with febrile symptoms in children."

The Society's investigations fail to establish an anatomical or clinical basis for a distinction between membranous croup and diphtheria.

The question of the possibility of exciting a membranous inflammation of the air passages through mechanical or chemical irritation may be appropriately considered in connection with the above report. If a false membrane can be produced in this manner, and on examination is found to be composed of histological elements distinct from those which form diphtheritic exudations, it would constitute an argument in favor of the existence of a membranous laryngeal affection, non-diphtheritic in character. Numerous experimental investigations have been undertaken during the past few years, with the view of settling these points. The results obtained by different observers are conflicting, and the question still remains *sub judice.*

Mayer's conclusions, based upon numerous experiments on animals, are as follows:

1. When ammonia is applied to the trachea of animals, the result is not completely identical with croupous inflammation in the human being.

2. The application of a small quantity of a weak solution of ammonia to the trachea and larynx does not produce any change in the histological structure of the superficial layers of the mucous membrane; it simply gives rise to a more or less severe catarrhal inflammation.

3. A larger quantity of a stronger solution acts like many other chemical escharotics, *i. e.*, a swelling and destruction of the superficial cellular elements is produced; an active cell proliferation taking place beneath the dead tissue assists in its removal.

In the lungs the ammonia gives rise to intense catarrhal inflammation.

Oertel, in his monograph on "inflammatory croup," vehemently denies the correctness of *Mayer's* observations. He asserts that he was able to produce in rabbits, by means of ammonia in solutions of varying strength, false membranes which were physically, chemically, and histologically identical with those called croupous membranes in the human being. The examination of a laryngeal pseudo-membrane apparently produced by cologne water, by the committee of the British Medical and Chirurgical Society, showed that only in a single layer was the exudation strictly comparable with a diphtheritic membrane removed from a similar situation. In the one case, however, sections were made of the larynx with the membrane adhering; in the other, merely sections of a cast thrown off during life. In membranous laryngitis of the human being apparently produced by mechanical or chemical irritants, the possibility of the *co-existence* of diphtheritic infection must always be entertained; particularly must it be considered in hospital practice, which furnishes the majority of such cases.

The non-infectiousness (?) of "membranous croup," as well as its sporadic character, have been advanced as arguments in favor of its purely inflammatory character. *Jacobi*, of New York, has suggested explanations of these apparent anomalies, which are well worthy of consideration. "Where muciparous follicles abound, their normal secretion, as a rule, prevents deep-seated degeneration of the tissue. Epithelial conglomerations are lifted up from the surface, the tissue takes little part in the morbid process, while the serum of the mucus penetrates the deposit and promotes its maceration." The numerous muciparous follicles of the larynx and trachea guard the lymph-ducts of the mucous membrane against the superjacent and loosened diphtheritic exudations, and hence constitutional infection is prevented.

He finds an explanation of cases of sporadic croup in the anatomy of the vocal cords. It may be summarized as follows: Inasmuch as the vocal cords form the borders of the narrowest entrance to the lungs, foreign bodies, whether of a benign or malignant character, will be retained by them. Their epithelium is of the pavement variety, which is principally concerned in the formation of the diphtheritic exudation. They have no muciparous glands or lymph-vessels; hence while a speedy removal of the diphtheritic deposit is impossible, at the same time general infection is prevented. "Where there is not poison enough for a thorough infection, there is still sufficient for a local deposit." "Where diphtheria has died out as an epidemic, the stray cases, with limited infecting power, will be known as so-called sporadic membranous croup."

In recent discussions of the relations of "membranous croup" and diphtheria, it is a significant fact that the opponents of the theory of their identity, while asserting the prevalence of a membranous affection of the larynx of a simple inflammatory character in the past, at the same time very generally admit that membranous croup as it occurs at the present time is evidently of a diphtheritic character.

In conclusion, it may be fairly claimed that an impartial study of recent pathological investigations and clinical researches points to the *identity of membranous laryngitis* ("*croup*") *and laryngeal diphtheria.* The varieties of croup originally suggested by *Bretonneau* accord with the above view. They are: 1. Spasmodic croup—Syn., Laryngismus stridulus. 2. Inflammatory croup—Syn., Simple or catarrhal laryngitis. 3. Membranous croup—Syn., Diphtheritic croup.

TREATMENT OF DIPHTHERIA.

The different methods adopted at the present time in the treatment of diphtheria are chiefly based upon the respective theories of its primarily local or constitutional origin. Whichever view is accepted, the acknowledged contagious properties of diphtheritic false membranes, and the danger of secondary infection * from their early decomposition, has secured for local medication a prominent position. In the use of many remedies employed for this purpose, we are also fulfilling the indications for a constitutional treatment. Local remedies may be conveniently divided into three classes: 1st. Those which dissolve the false membranes and facilitate their removal. 2d. Those given with the intent of combating the inflammatory reaction of the mucous membrane. 3d. Disinfectants.

The agents of the first class, which are still considered most efficient, embrace hot vapor, lactic acid, lime-water, carbonate of lime, and glycerin. *Oertel's* claim of the almost specific action of hot vapor, in not only hastening the separation of the false membranes, but in also arresting the inflammatory reaction of the mucous membranes, does not seem to have been borne out by clinical experience. Against its prolonged use the objection has been urged that, while undoubtedly softening and facilitating the separation of the false membranes, it at the same time softens healthy tissue and thus permits of the deeper penetration of the poison. The reputed value of lactic acid and the lime preparations as solvents of diphtheritic membranes has been in a measure confirmed. Inhalation of a one-fifth per cent solution of the former, in cases of laryngeal diphtheria, *is* still earnestly advised. To be efficient they should be repeated at very frequent intervals. The remedies of the second class, which continue to hold a prominent place in the therapeutics of diphtheria, are chlorate of potash and the perchloride of iron. The use of the former, in large doses, in diphtheria, was introduced by *Vogel*, in 1860; the treatment by the perchloride of iron, by *Gigot*, in France, in 1858. The value of the latter salt, both as a local and constitutional remedy in diphtheria, has received the very general confirmation of clinical experience. Its probable action as an ozonizing agent in the blood, its properties as a disinfectant, its apparent power, shared with the other iron salts, of acting upon the vital contractility of the tissues, and finally its well-known value as a stimulant of the nervous system, have been suggested in explanation of its efficiency. To Dr. *Jacobi's* exhaustive monographs on the subject, supplemented by the observations of Dr. *Billington*, is largely due its very general adoption in the treatment of diphtheria by American practitioners. It cannot be too earnestly insisted upon that to secure its full effect it must be given in large doses frequently repeated. Dr. *Jacobi* claims that only in doses of five to fifteen drops of the tincture, every hour or half-hour, is its efficacy fairly tested. A combination of chlorate of potash and the tincture of the perchloride of iron, in the proportion of two grains of the former to five drops of the latter, in equal parts of glycerin and water, repeated at intervals of a half-hour, is one much used by American practitioners, and which well fulfils the indications of both a local and constitutional medication. The third class embraces a very numerous list of agents, among which carbolic acid, the bromine preparations, salicylic acid, hydrate of chloral, benzoate of soda, benzoic acid, quinine, and sulphur are most favorably known at present.

---

* The term secondary infection is used in this article in the sense of infection due to the absorption of the products of putrefaction.

The disinfecting power of carbolic acid and its value as a local remedy in diphtheria need not be dwelt upon. *Letzerich's* experiments would seem to prove that salicylic acid, in the proportion of one to sixty, is capable of instantaneously destroying diphtheritic micro-organisms. Solutions of salicylic acid of this and greater strength have been used, as local applications, with decided benefit. I have obtained excellent results from insufflations of the powdered acid combined with bismuth, in the proportion of one to six or seven. The Italian physician *Ferrini* first suggested the local use of chloral -hydrate as an antiseptic in diphtheria. His method is to paint the diseased surfaces with a solution of chloral hydrate and glycerin, in the proportion of one to six, three or four times daily. It is claimed to be an efficient remedy. Mr. *Graham Brown's* experiments in Prof. *Klebs'* laboratory for testing the comparative efficacy of hydrochlorate of quinine, salicylate of soda, and benzoate of soda in destroying the contagious principle of diphtheria assign the first place to the last-mentioned salt. Mr. *Brown* suggests hypodermic injections of benzoate of soda as a prophylactic against diphtheria. *Klebs* claims that, for the exercise of a specific action, it must be given in very large doses. A drachm and a half may be given daily to children of five years of age, in frequently repeated doses, combined with insufflations. From the known antiseptic properties of quinine, solutions and powdered preparations of the alkaloid have been used as topical applications. The power of quinine as a *disinfectant of living blood* would seem to have been established by the elaborate experimental researches of *Binz* and *Kerner*. Its efficacy in combating sepsis in the human being is an indication for its internal use in the secondary infection of diphtheria. When given internally, in large doses, the unirritating preparation of the hydrochlorate should be used rather than the acid sulphate.

In regard to the efficiency of sulphur as a local application, *Oertel* asserts, as the result of numerous experiments, that its action is purely mechanical. Where the exudation has already ceased, it acts as a scouring powder, wearing off by friction the membranes which are already loosened. Under such circumstances its utility is undoubted. Inhalations of the oil of eucalyptus have of late been recommended in diphtheria. Its use is based upon its antiseptic properties, which it possesses with all aromatics, and its slightly stimulating and astringent action. A formula which has been advised is as follows:

℞ Ol. eucalypt. fol.	ʒ ss.
Spt. vini rectif	ʒ v.
Aquæ destill	℥ vi.

M. S. To be employed for ten inhalations, at intervals of an hour.

In accordance with the experimental evidence adduced by *Nicati*, that the extension of the diphtheritic membrane is checked by excluding the oxygen of the air, the advisability of coating such membranes, on their first appearance, with an ethereal tincture of gum tolu or some other similar preparation has been suggested.

Local Treatment in Nasal Diphtheria.—In no form of diphtheria is the benefit to be derived from local treatment more conspicuous than in that attacking the nasal cavity.

Through the facilities offered for rapid decomposition of the false membranes and absorption, it has justly been considered of the gravest import. Yet clinical experience of the past few years has taught that, of all the forms of the disease, nasal diphtheria is the most easily controlled by appropriate treatment. With the earliest appearance of the disease in the nasal passages, frequent syringings should be employed. They may be repeated at intervals of two hours, an hour, or even a half-hour in grave cases. A three- or four-grain solution of carbolic or salicylic acid will fulfil all indications. If the nostrils are plugged with false membranes and secretions, they may be first cleansed by means of syringing or spraying with five or ten grain solutions of carbonate of soda or borax, and the disinfecting remedies applied after a free passage has been secured. An ordinary hard-rubber ear-syringe is more easily used in children than the douche. By this method of treatment, faithfully carried out, the disease rapidly disappears from the nasal cavity, the swelling of the neighboring glands subsides, and an imminent danger is averted.

GENERAL TREATMENT.

The general treatment of diphtheria has been partially considered in the foregoing remarks upon the use of local medication. However energetic may be the employment of the latter, clinical experience has taught that it must be combined with a tonic and restorative treatment. Little need be added on this subject. The principles are the same as those which form the basis of the treatment of other infectious diseases. In cases of diphtheria where the necessary alimentation cannot be secured by the natural channels, I would suggest the use of enemata of defibrinated blood.* The method has been fully described by Dr. *Andrew H. Smith*, of New York, and has been used by him and others with gratifying results. It has seemed to me to be a more perfect aliment and one producing a more decided revivifying effect than the ordinary preparations of milk and egg, *Leube's* extract of meat, etc., administered in a similar way. In conclusion, a word may be added in regard to the internal use of alcohol in the treatment of diphtheria. Doubts may exist as to its utility in mild cases of the disease; in cases in which the symptoms of a secondary infection are present, its value cannot be overestimated. *Sanné* regards it as the most potent of all internal antiseptics in diphtheria. *Jacobi* has expressed a nearly similar opinion. I have given six to twelve ounces of brandy in the course of twenty-four hours, to children of three and four years of age, exhibiting the most pronounced symptoms of sepsis, and have thereby, I believe, saved life.

---

*Two ounces of defibrinated blood can be given in the form of enemata twice daily to children of four or five years of age. The preparation is well known to butchers under the name of "stirred" blood.

TRACHEOTOMY IN MEMBRANOUS LARYNGITIS.

A brief résumé of prevailing views, in regard to the advisability of the operation of tracheotomy in cases of membranous laryngitis, with a reference to the most recent statistics on the subject, will alone be attempted in the present article.

Jacobi, of New York, in *Gerhardt's* Hand Book of Diseases of Children (1877), in referring to his 67 cases of tracheotomy in laryngeal diphtheria, published in 1868, of which 13 recovered, states that the mortality in his subsequent 100 cases has so reduced this percentage that at the present time he operates only to avoid the distressing scene of death by suffocation. *Oertel*, in the second edition of *v. Ziemssen's* Cyclopædia, expresses an equally unfavorable opinion. Dr. *J. Solis Cohen*, of Philadelphia, in an exhaustive monograph on "Croup in its Relations to Tracheotomy" (1874), gives the following statistics, derived from American sources: number of tracheotomies, 325; recoveries, 84.

Mr. *Parker's* statistics, published in the British Medico-Chirurgical Transactions (1879), are exceedingly favorable. His success has been so exceptional that a description of his method of operation and after-treatment may prove instructive. He regards the recession of the chest-walls and supra-clavicular spaces, especially in conjunction with a more or less complete suppression of voice, and labored *expiration*, as indications for an immediate operation. He advises the use of an anæsthetic, as a rule, and recommends the high operation, in reality crico-tracheotomy. He thoroughly exposes the trachea before it is incised, as this procedure permits of the opening being made with greater precision, and also facilitates the introduction of the canula; he believes that it, moreover, diminishes the danger of subsequent infiltration of pus into the muscles and intramuscular planes. The methodical use of a dilator is urged after the incision of the trachea, and also the passage of a large feather downwards into the trachea and upwards towards the glottis, so as to detach all the membrane possible. He suggests that the presence of membrane and inspissated mucus above the tube, after tracheotomy, is a frequent though unsuspected source of reflex cough and irritation. Mr. *Parker* favors the use of as large a tube as can be introduced into the trachea without violence, and as short a one as is consistent with safety. Much importance is attached to the curve of the canula, and one which corresponds more exactly with the direction of the trachea than the rigid tube in common use, is proposed. According to Mr. *Parker*, a solution of carbonate of soda (10–20 grains to an ounce of distilled water), used as a spray, in conjunction with steam, is the most effectual means of liquefying and facilitating the removal of membrane. A free secretion from the trachea he regards as a not unfavorable sign; on the contrary, a "dry tracheotomy" is always a source of anxiety to him. He recommends the very frequent and careful cleansing of the inner tube, and the withdrawal and cleansing of the outer one after the lapse of twenty-four or thirty-six hours. In conclusion, he urges the vital importance of the continued treatment of the *disease*, of which the membranous exudation is a symptom. Out of twenty-one cases of membranous laryngitis, treated according to the above method, there were twelve recoveries.

In a recent paper by Mr. *George Buchanan*, of Glasgow, fifty tracheotomies in "croup" and diphtheria (personal cases) are reported, with nineteen recoveries. Recent statistics, derived from French sources, are as follows:—In the Hôpital Sainte Eugénie (1854–1876), there were 2312 tracheotomies performed in cases of laryngeal diphtheria, with 509 recoveries; in the Hôpital des Enfants Malades (1851–1876), there were 2351 operations, with 614 recoveries.

The reports of successful individual operations scattered through the medical literature of the past few years afford further convincing proof of the service which has been rendered by tracheotomy in membranous laryngitis.

Indications for Tracheotomy.—The prevailing French practice is to operate as soon as the asphyxia becomes *continuous*. *Paroxysmal* attacks of suffocation, however, can only be safely disregarded under circumstances which permit of the constant attendance of the surgeon. Among English surgeons, the precise indications for operating find expression in the views of Mr. *Parker*. Tracheotomy is contra-indicated in those cases alone in which the asphyxia is due to an extension of the disease into the smaller bronchi, a condition which presents many difficulties for a positive diagnosis. Mr. *George Buchanan* has suggested the observance of certain physical signs to determine this point. Recession of the chest-walls will occur when the obstruction is confined to the larynx and trachea; on the other hand, an implication of the smaller bronchial tubes will give rise to a condition of the chest resembling that seen in emphysema, *i. e.*, the movements of the chest-walls are impeded, the whole chest being lifted in inspiration, as if composed of one piece.

A more exact observance of the indications for surgical interference in membranous laryngitis, which may again be stated as consisting in a more or less complete suppression of voice, a recession of the chest-walls in inspiration, and an obstructed expiration, together with a most careful attention to after-treatment, both local and general, may be regarded as alone promising greater success than has yet been attained by the operation of tracheotomy in laryngeal diphtheria.

VARICELLA.—MEASLES.—RICKETS.

BY

F. FORCHHEIMER, M.D.,

OF CINCINNATI,

PROF. OF PHYSIOLOGY AND CLIN PROF. OF DISEASES OF CHILDREN, MED. COLL. OF OHIO.

VARICELLA.

BIBLIOGRAPHY.

Henoch: Berl. klin. Wochenschrift, 1874, p. 211.—**Senator**: Jhrbch. f. Kinderhlknde., N. F., VII., p. 444, 1874.—**Hagenbach**: Jahresb. üb. d. Kinderspital zu Basel, 1874, p. 44.—**Reimer**: Petersb. Ztschr., N. F., V., p. 251, 1875.—**Wehner**: Diss., Würzb., 1875.—**Kaposi**: Archiv f. Dermatologie u. Syph., II., p. 255, 1873. —**Gamberini**: Archives of Dermatology, Vol. I., p. 69, 1874-5.—**Hutchinson, Jonathan**: Brit. Med. Jour., pp. 631 and 663, 1875.—**Edis**: Brit. Med. Jour., p. 702, 1875.—**Filatow**: Eine Varicellen-Epidemie im Mosk. Kinderspital. Mosk. med. Gaz., 1877, and Centralzeitung f. Kinderheilkunde, p. 371, 1877.—**Bohn**: Gerhard's Handbuch, II., 325, 1877. Buffalo Med. and Surg. Jour., 1877-8, p. 228.

In the years intervening between the appearance of the first German edition of *v. Ziemssen* and to-day, the war between the identists and the dualists has still been continued. On the one hand, we have the dermatologists, *Hebra* and his followers, and on the other, the children's physicians; the one claiming that the poisons of variola and varicella are identical; the other, that they are distinct and separate. *Hebra* and his followers base their opinions upon the following: that the eruption is the same in both diseases; that both may or may not leave cicatrices; that they occur at the same time and in the same epidemic; and that the one may give rise to the other. Each one of these points has been taken up by those making a specialty of children's diseases, and answers have been filed. In regard to the identity of the eruption it is claimed that in varicella we have a vesicle whose contents may become turbid, but not made up of pus alone; that this vesicle does not rest upon an infiltrated surface as in small-pox (which in the latter disease gives the impression of "shot" when felt); that there is no umbilication of the vesicle; and finally, that cicatrices are the exception in varicella.

In addition, it is claimed that the whole aspect of the patient with small-pox differs from that of the patient with chicken-pox. In the latter disease, we have the eruption irregularly scattered over the body, beginning usually somewhere on the upper half of the body; but its characteristic is, that upon the face we may see vesicles, while upon the chest we already have crusts, or vice versa, or that next to a vesicle in full bloom we have one upon whose centre the crust already begins to form, and next to this, one that is already covered with a crust and where the areola has entirely disappeared. In small-pox, the picture is entirely different, the eruption follows a given rule, and upon the same parts of the body we usually have the same character of eruption. Although all this has not been denied, yet the dermatologists claim that cases of variola do exist in which these symptoms are present, and some dualists (*Senator*) admit it freely, so that, although typical cases are considered as essentially differing from each other, yet cases do exist in which there seems to be a running together of the external appearances. The dualists lay principal stress upon the proof that the poisons are different, and this has been especially well done by *Senator*, whose line of argument is briefly given here. In regard to this eruption appearing at the same time with variola, *Senator* claims that varicella does appear as an independent disease in the form of epidemics, and cites instances in which this has taken place. *Kaposi*, in answer to this, states that the poison of variola has been lurking about, and its action cannot be excluded by stating that no cases of variola exist at the time of the breaking out of the chicken-pox epidemic. In answer to this, *Senator* asks why it is that this peculiar variola poison attacks children only, and produces varicella, and why we never have severe cases of variola produced as a result of infection with varicella poison, as we do have varioloid produce the most violent forms of small-pox.

Lastly, *Senator* claims that varicella and variola do not have any influence over each other; in other words, that a patient who has had varicella can get small-pox just as if he had never had varicella, and the converse. This is admitted, and therefore the identists must state that when a patient has had varicella and then gets small-pox he has had small-pox twice. That patients have small-pox twice is known to occur; yet this is very rare, three cases only being recorded, and they, as exceptions only, going to prove the rule. The opposite can be said of the occurrence of varicella and small-pox in the same individual, namely, if a patient has had small-pox and then is exposed to varicella he will be sure to get the latter disease, provided he is not too old. Furthermore, vaccination protects against variola, but has no power over varicella. *Senator* relates the following cases in this connection: a child having been successfully vaccinated, was seized with varicella eleven days after vaccination; three others, in two of which vaccination was successful, and in the third revaccination unsuccessful after an attack of varicella. Furthermore, he records the case of a child that had had varicella twice and then was successfully vaccinated. He then relates another case in which a child that had had small-pox in 1868 was unsuccessfully vaccinated in 1869 and 1870, then was taken with varicella and again frequently vaccinated in 1871 without its taking.

In connection with this, a case of varicella is narrated as occurring in St. Thomas' Hospital, in which in a child variola appeared, while the scabs of varicella were still distinctly present upon the patient.

Steiner has arrived at positive results in the inoculation of varicella, and by means of these has determined the period of incubation as lasting eight days.

In regard to the symptoms, discussion still exists concerning the prodromal fever, *Thomas* asserting that this does not exist as a rule—a fact which was also observed by *Filatow* in the epidemic described by him. *Senator* considers that it does, and in this is supported by *Bohn.*

Mr. *Jonathan Hutchinson* describes a new disease which he calls varicella-prurigo, the peculiarities of which are as follows: it follows either chicken-pox or vaccination, its duration is seldom less than months, and it is not amenable to treatment. To quote from the author, "their (these cases) peculiarity consists in that the eruption (*i. e.*, the eruption of varicella), instead of disappearing in a few days, is indefinitely prolonged by the succession of fresh crops, and that the spots ulcerate and scab, sometimes becoming large sores. Great irritation is produced, and the child becomes fretful and thin. The eruption may last for months, and the spots then come to resemble ecthyma on the one hand, or, yet more frequently, the disease known as "lichen urticatus." The eruption, it is claimed, after the first stage does no longer resemble that of varicella, but becomes vesicular or bullous, papular or pustular. Either one or all of these forms of eruption may be present, and they are situated upon the feet, the hands, the legs, the arms, and the head. Most commonly are found vesicles or bullæ on the soles of the feet and palms of the hands and scabs on the head (porrigo). As the disease progresses, the itching and the consequent scratching produce their effects, and the patient is covered with scratch-marks and their sequelæ.

Mr. *Hutchinson* reports twenty-eight cases, sixteen following varicella, and twelve vaccination, the youngest being six months and the oldest eight years of age. When first attacked, the youngest was six weeks old, and the oldest seven years. At the time of admission, the disease had lasted from two weeks to two and one-quarter years. According to the author, "only in a minority anything approaching a cure was effected." Several explanations are offered by the author: first, that we are dealing with a prolonged form with pruritus, and secondly, "that the outbreak of varicella in some way affects the nerve-structure of the skin as to induce a state of pruriginous irritability." Mr. *Hutchinson,* however, believes that the same process in the skin that produces varicella also produces this disease, and sums up as follows: "That varicella, varioloid, the rash which sometimes attends vaccination, and possibly other exanthems possess the power, in exceptional cases, of making the skin irritable, and thus laying the foundation for long-continued and most troublesome conditions of prurigo," and furthermore, "that this consequence is especially apt to ensue when, as is not infrequent in varicella, the eruption is long protracted, and occurs in successive crops."

Mr. *Balmanno Squire,* on the other hand, claims that this disease has no existence *eo ipso,* but is simply ecthyma or "lichen urticatus." He bases his views upon the fact that the varicella element of the disease was not observed by Mr. *Hutchinson,* but was taken for granted by him on account of descriptions or statements given by other parties, and furthermore, that the description given by Mr. *Hutchinson* corresponds to the diseases mentioned by him. It may be mentioned here that one case (*Edis*) is on record in which the varicella was observed by the physician and reported by him as a case identical with those observed by Mr. *Hutchinson*. Inasmuch as the description comes from such high authority and is objected to by one whose name is so well known to dermatologists, it is to be hoped that the future will disclose whether this actually is a newly discovered disease; and we venture to hope that if this should prove to be the case it shall be given a better name than varicella-prurigo.

MEASLES.

BIBLIOGRAPHY.

Fleischmann: Archiv f. Derm., 1872, IV., p. 223.—v. Guttceit: 30 Jahre Praxis, I., p. 248.—Scheby-Buch: Arch. f. Derm., V., 1873, p. 246.—Coyne: Virchow-Hirsch, J. B., 1874.—Demme: 12. Med. Ber. üb. d. Kinderspital zu Bern, 1874, Bern, 1875, p. 6.—Czanderna: Med. Chirurg. Centralblatt.—Faye: Ctrlztg., 1874, p. 659.—Kassowitz: Oest. Jhrbch. f. Päd., 1874, V., p. 79.—Lorey: Frankfter. Jber., 1870, XIV., p. 151; 1871, XV., p. 199; 1874, XVIII., p. 132.—Modestin: Wien. med. Wch., 1874, p. 838.—Oppenheim: Oest. Jhrbch. f. Päd., 1874, V., p. 93.—Steiner: Jbch. f. Kdhknde., 1874, VII., p. 346.—Boese: Allg. med. Ctrlztg., 1875, p. 95.—Dunbar-Tweedale: Ctrlztg., 1875, p. 451.—Gassot: V.-H. Jber., 1874, II., p. 78.—Hennig: Jbch. f. Kdhlknde., 1875, VIII., p. 417.—Henoch: Ctlztg., 1875, p. 828.—Hesse: Diss. quoted in Berl. kl. Wchschrift., 1875, p. 535.—Jenner: Ctrztg., 1875, p. 915.—Königer: Berl. kl. Wch., 1875, p. 610.—Lewinski: Berl. kl. Wch., 1875, p. 493.—Müller: Union méd., 1875, p. 387.—Rauchfuss: Pet. med. Ztschr., N. F., 1875, V., p. 278.—Stage: V.-H. Jber., 1874, II., p. 78. Brit. Med. Jour., 1876.—Cordier: Jbch. f. Kdhkde., IX., p. 412.—Hagenbach: Jbch. f. Kdhkde., 1876, IX., p. 56.—Kerning: St. Pet. med. Wch., 1876, 14.—Klebs: Würzbr. Verh., N. F., V., 1874.—Kugelmann: Vortrag üb. Verminderung d. Sterblichkeit, Hanover, 1876.—Leopardi: Ctrlbl., 1876, p. 380.—Reimer: Jbch. f. Kinderheilkunde, 1876, X.—Schmidt-Rimpler: Berl. kl. Wch., 1876, p. 193.—Steinitz: Ctztg , 1876, p. 409.—Taube: Beitr. z. path. Anat. d. Masern, Leipzig, 1876.—Tripe: Jbch. f. Kdh., 1876, IX., p. 411 (section for public medicine, Brit. Med. Association, 1875).—Bohn: Gerhardt's Handbuch d. Kderkkhten., II., p. 295, 1877.—Baginsky: Oest. Jbch. f. Päd., VIII., V. II.—Baginsky: Centralzeitung f. Kinderheilkunde, 355, I., 1877.—Förster: Einige Bemerkungen üb. d. Verbreitungsweise d. M. u. d. Sch., Jbch. f. Kdhlknde., X., 152.—Squire: Measles in Fiji, Br. Med. Jour., 1877.—Braidwood: Med. Times and Gaz., 1878, p. 573.—Fox, H. Courtenay: Med. Times and Gaz., 1878, p. 587.—Czanderna:—Med. Chirurg. Centralbl.; Centralzeitung f. Knderhlknde., No. 14, 1879.—Sidlo: Vortrag, Wien, 1879.—Alsberg: Archiv f. Kinderheilkunde, I., 708, p. 255, 1880.

Since the days of Hallier and Salisbury, little has been done to determine the exact nature of the contagium of measles. Unfortunately, these two observers, although claiming to have obtained positive results, have not found these verified by others, so that observations in this direction must be begun anew. *Klebs* reports as follows regarding his results with the micrococci of measles: he obtained them from the trachea and from blood taken from the hearts of infant cadavers. In the latter, collected in flattened capillary tubes, there developed balls of micrococci; in the trachea, both micrococci and bacteria were present in large quantities. Under observation, pale, finely-granular micrococcus balls developed, and changed very quickly to bacteria, which moved about very actively. These sought the periphery, about 1–2 mm. distant from the centre of development, and formed a zone, comparable with a hedge or fence that is composed of rods. From this there were formed new masses of micrococci, but further no regular process of arrangement or development could be observed. *Braidwood* has also described a bacterium which he claims to have seen in the moisture collected from the breath, in the lungs, and the liver of measles patients. He describes them simply as "sparkling bodies," which seem to differ considerably from those described by *Klebs.*

In regard to mortality from measles and the influence of sex and age, Mr. *H. Courtenay Fox* has collected very valuable material, in the form of statistics, which certainly, for England and London, are conclusive. He states that the mortality from measles, in England, is 439 per million, and in London, 567; that measles destroy one-half as many lives as scarlatina. The ratio of the two sexes is as follows:

	MALES.	FEMALES.
England	457	422
London	620	522

Mr. *Fox* specifies by giving the "mortality of males and females at specific ages (mean annual deaths out of one million of each sex, living at each age)" as follows:

	ENGLAND.		LONDON.	
	Males.	Females.	Males.	Females.
Under one year	3022	2530	3571	2987
One and under two years	6086	5825	8630	8050
Two " three "	3178	3255	4683	4757
Three " four "	1730	1851	2594	2620
Four " five "	980	1028	1358	1466
Five and under ten years	255	278	301	316
Ten " fifteen years	29	38	24	32
Fifteen " twenty "	9	13	9	11
Twenty " twenty-five years	7	9	5	7
Twenty-five and under thirty-five years	5	8	5	7
Thirty-five " forty-five "	3	5	2	3

It will be seen from these statistics that males are more frequently attacked than females, a statement frequently made, but just as frequently denied by authorities. *Thomas* comes to the conclusion that sex has no influence upon the predisposition to this disease. The age that is most subject to measles is between one and two years. The most remarkable fact that is proven by these statistics is, that so many children under one year of age are attacked, being nearly as many as between two and three years, and more than between three and four. Taking into consideration the immunity that infants at the breast are supposed to possess, the age between six months and one year seems to be especially liable to this disease.

The fact that measles, when coming among uncivilized people, or when attacking a country that has long been free from the disease, is a very dangerous affection, has again been demonstrated in the Fiji islands. It was brought to the islands (1877) from Sydney, and in a comparatively short time caused twenty thousand deaths, being from one-fifth to one-fourth of the entire population.

Steiner has reported four very interesting cases of what he terms *morbilli bullosi sive pemphigoidei.* In these four cases, of which one died of acute hydrocephalus and pneumonia, all occurring in the same family, there appeared, in addition to the regular eruption of measles, large vesicles filled with a turbid fluid. The vesicles were found upon the face, back, breast, external genitals, hands and feet, and in the nose and mouth. The eruption did not appear at the same time in all the cases, and when one crop had disappeared, another would develop. Nothing was found in the family history to account for this peculiarity, nor had any of the children ever suffered with pemphigus before. *Steiner* states that, in six thousand cases of measles that had come under his observation, these are the only cases of this description that he has seen.

Pathological changes that were peculiar to one epidemic were observed by Dr. *Taube* (*Thomas*). In some of his cases, the lungs were affected to such an extent that respiration became a mechanical impossibility, this being due to the great amount of infiltration present. On account of this infiltration, the lobular nature of the pneumonia was somewhat effaced, and the whole assumed the character of the phthisical process of adults, except that this result had been produced in weeks. The lungs contained cheesy masses, sometimes confluent to such an extent as to involve a whole lung. Not infrequently these were found broken down, to have formed cavities varying in size, and, in one case, to have produced a fatal hemorrhage. The larger bronchial tubes of the parts thus affected were found softer than normal, their mucous membrane reddened and swollen, their contents a white mucus, which was sometimes fetid. Dilatations, cylindrical and sack-like, of the walls of the bronchi were observed frequently. The smaller bronchi were thickened by peri-bronchitis, so that their walls became inelastic and of the color of the surrounding parenchyma of the lungs. True miliary tubercle could not be discovered in any of these cases. The cheesy degeneration was least observable in those lungs that had been compressed by copious pleuritic exudations, but even here it was present. Microscopically, the greatest amount of change was observable in the spaces between the capillaries and the epithelium of the lungs; they were dilated and filled by cells which partook of the nature of epithelium, smaller than the epithelium of the alveoli, but larger than white blood-corpuscles. The alveoli were found to contain cells, but this was not the cause of the solidification, as there always was present in the alveolus a space not filled by cells. The origin of these peribronchial and perivascular cells was detected by *Taube* to lie in the mucous glands of the bronchi, and the whole process, called by him *catarrhal interstitial pneumonia*, is to be considered as an adenitis of the bronchial mucous glands (*Thomas*). When these glands were examined, the changes could be observed from the simple proliferation of the cells lining the follicle, to its complete filling up. But these cells do not undergo their physiological change, mucous metamorphosis; they remain as they are, penetrating the basement membrane of the gland, proliferating into the tissues, and finally into the inter-alveolar spaces, producing the changes above described.

The more closely cases have been observed, the more lesions are detected, not only in the skin, as has been shown above, but also in other organs. Thus, in connection with measles, diseased conditions of the eye, the intestinal tract, the heart, the kidneys, the liver, the spleen and the tonsils have been observed and accurately described. It has been shown that, in some epidemics, the tonsils enlarge, and then become smaller as the eruption recedes, so that, in some cases, the differential diagnosis between measles and scarlatina might become difficult, especially when a true parenchymatous nephritis, with dropsy, follows as a sequela. Infants, although they may not be affected by the measles as a disease, may succumb to the catarrhal affection of the intestinal tract, so that this ought always to attract the attention of the careful physician.

In connection with the treatment of this disease, nothing new has been developed. The antipyretic and antithermic methods have gained ground, and with them the older views of the danger of "driving in" the eruption and of catching cold are daily losing force. At the present, nobody is afraid to give a patient with high temperature a bath, nor does he expect it to be followed by a pneumonia because the eruption is "driven in." Fresh air in abundance is given to these patients, and yet our mortality from this disease is not as great as it was before the days of the thermometer.

RICKETS.

BIBLIOGRAPHY.

Fleischmann: Ueber d. angeblichen typischen Verlauf d. Rachitis. Jahrb. f. Kdhlknde., 1874, p. 413.—L. **Tripier**: Deschambres' Dictionnaire des Sciences med., IIIme ser., I., 1874, and Archives de physiol. norm. et pathol., 1874, p. 108. —**English**: Ein Fall von R. fœtalis. Oest. Jahrb. f. Pädiatr., 1874.—**Woronichin**: Ueber d. Einfluss d. Körperbaues, d. Ernährung u. d. rhachit. Prozesses auf d.

Durchbruch d. Milchzähne. Jahrb. d. Kinderheilkunde, IX., pp. 91–105.—Percheron: Gaz. hebdom., No. 30, 1875.—Roloff: Ueber Osteom. u. Rhach. Gerlach's Arch. f. wiss. u. prakt. Thierheilk., 1875, I., p. 189.—Habner: *a.* Rhach. u. Tuberculose beim Rinde nach Hüttenrauchfutter. Jahresb. d. Dresd. G. f. Naturhlknde., 1875–6; *b.* Ueber Erzeugung u. Verhütung d. R., ib., p. 125. —Bernard (de Montbrun): De la cure du rachitisme par le lait de chienne. Gaz. hebdom., 1876, No. 2.—Toussaint et Trepier: Sur les effets de l'acide lactique etc. Bouley's Recueil de méd. veterin., 1876, III., p. 750.—Rehn: Centralzeitung f. Kinderheilkunde, I., p. 227, 1877-78.—Bradley: Lancet, July 21st, 1877.—Th. Jones: Lancet, vol. 2, p. 235.—Senator: V. Ziemssen's Handbuch, Vol. XIII., p. 178–229.—Fleischmann: Ueber Kiefer-rhachitis. W. med. Presse, 1877. Klinik d. Pädiatrie, 1877, II., pp. 158–187.—Forchheimer: Thirteen Cases of Rachitis. The Clinic, 1877.—Spiegelberg: Lehrbuch d. Geburtshülfe, pp. 356–358, 1878.—Dauvergne, Père: Des causes et du traitement du Rachitisme, 1878.—Stockfleth: Knochenversuche b. Ferkeln u. Laufschweinen. Deutsche Ztschr. f. Thiermed., etc., 1878, IV.—Eberth: Die Fœtale Rhachitis, etc., 1878. —Rehn: Gerhardt's Hdbch. d. Kkhten., III., 1 H., 1878. Centralztng. f. Kinderheilkunde, II., p. 83, '78–79.—Forster: Ueber d. Verarmung d. Körpers an Kalk. Ztschr. f. Biologie, XII., p. 464.—Wyss: Discussion, Jahrb. f. Kinderheilkunde, N. F., XIV., p. 380.

With every year the subject of rachitis is receiving more attention from the physicians of this country. The reasons for this are many, but the most important is undoubtedly the fact that, with us, the disease is absolutely on the increase. This, in its turn, is due to the growing city population; to the tendency to centralization, and, with it, all the baneful causes of diseases consequent upon overcrowding, city diet and food, poor air, etc. Statistics, unfortunately, are not at hand to prove this assertion; but from verbal communications from physicians practising in the west, and from my own experience, it is beyond doubt that for regions that were comparatively thinly settled fifteen or twenty years ago this statement is correct. Thus, in my clinic, rachitis has increased, within the last five years, nearly twenty-five per cent. Another reason is, that the disease is beginning to be more thoroughly understood, and its importance as a factor in infant mortality better appreciated. It is true, that the more violent forms of this disease are comparatively rare among the white population of this country, but the milder forms occur frequently, and although not sufficiently severe to cause uneasiness *per se*, yet they often suffice to act as causative elements in the production of concomitant diseases, and are therefore sufficiently important to merit close study.

In regard to the age of rachitic patients, the latest authorities seem to be positive that both fœtal and congenital forms occur (*English*, *Rehn*, *Senator*, *Spiegelberg*), but the data offered by *Ritter v. Rittershain's* statistics, that rachitis is most frequently developed during the first year of life, are continuing to be verified by careful observation. *Rehn* states that he never saw rachitis develop after the third year of life and statements like those of *Percheron*, in which a young man of twenty was supposed to have had an attack of rachitis, are only to be believed when accompanied by reports of microscopic examinations of the bones, presenting the characteristics of the disease.

Heredity, by some authors, is claimed as a positive cause for this disease. On the other hand, the line of argument is employed that neither pathology nor chemistry has furnished facts pointing to a rachitic diathesis; that rachitis does not depend upon any specific poison, but affects only certain well-known organs. The fact, however, that parents who have had rickets are more liable to have rachitic children than those not having had the disease (*Ritter*) is agreed to, and exception is only taken to the conclusions drawn from it. It is stated that a disease, having run its course twenty or more years before a woman becomes pregnant, a disease, moreover, not dependent upon a poison not eliminated from the body, could have no effect upon the fœtus. This may be true, but the deformed bones, and perhaps the process producing these, would certainly, according to the laws of heredity, be reflected in the offspring of parents possessing these. This can be verified in this country where the children of emigrants are nearly always rachitic, provided the parents have suffered in the same way. Cases have come under my observation in which the square head of the parents was retained in the children, without the production of any other symptoms of rickets, so that the form of the head had to be considered simply as the result of heredity.

The pathogenesis has received considerable attention, but the experimental facts upon which this is based are as yet so conflicting that nothing has been positively settled. The various theories may be classed under the following headings: first, the presence of too much acid in the blood; secondly, the absence of lime and its salts; thirdly, a combination of the two preceding with or without the consideration of some irritant. In regard to the first, the oldest, which had received a new impetus by the experiments of *Heitzmann*, it may be said that only one opinion exists. The results of *Heitzmann*, that rickets can be produced by the introduction into the system of large quantities of lactic acid, have not been corroborated by subsequent observers (*Heiss*, *Tripier*, and partially *Roloff*), so that this evidence can be practically set aside. *Senator* still claims that in rickets the solubility of the lime salts is influenced by the presence of acids in the blood, not, however, by acid blood which does not exist; he maintains this for both lactic and carbonic acids. The second view has found an able supporter in *Roloff*, who has produced rickets in growing animals simply by giving them food deficient in lime salts. He states that this is the sole cause of rachitis, leaving the question of the pathological changes, that precede the absence of calcification, entirely out of sight. Again, other experimenters have failed to produce rickets by this method (*Schütz*, *Weiske* and *Wildt*). *Wegner*, however, accounts for the proliferation which takes place in the cartilage by the introduction of some irritant into the blood. In his experiments he uses phosphorus as the irritant, and, at the same time, gives an insufficient amount of lime. Rachitis is always the result and can be proven by microscopic examination of the affected bones. It must be stated that, as yet, the experiments of *Wegner* have not been verified by any other observer. *Senator*, also, conceives of two elements in the production of rachitis; first a deficiency in lime, and secondly the presence of some irritant. The first, the deficiency in lime, is subdivided by him into two possible contingencies. He states that either the lime is not absorbed properly, or that the ossifying tissue does not have the property of taking up and retaining the lime salts. Either one or both of these factors may be at work. The occurrence of the first is proven by analyses of feces of rachitic children, in which the solid residue was present in as great a quantity as in adult stools. The second, *Senator* explains by referring to the presence of the phosphates in abnormally great quantities in the urine (denied by *Simon*, *Neubauer*), but, moreover, by conceiving that the blood has become diminished in alkalinity by the presence in it of lactic and carbonic acids. This, naturally, causes the phosphates to be dissolved in greater quantity than normal, and in this way prevents their becoming deposited in bone. The presence of lactic acid in the blood in abnormal quantities is caused by the fermentation of any or all the articles of food taken by the infant, and that this does occur the results of urinalysis verify. In three cases in which lactic acid was not found in the urine, *Senator* claims that it had already become converted to carbonic acid gas. For the increase of carbonic acid reasons are many: the increase in lactic acid, this, in its turn, being converted to carbonic acid, the increased formation of carbonic acid in inflamed tissues, and finally the disturbance caused by lesions in the respiratory organs. Carbonic acid has been found to be increased in rachitic bones. The second element in the production of this disease, the irritant, is considered by *Senator* to consist in increased growth of organs contained within bony cavities causing irritation of the bony walls, and then finally the using of bones, as in walking, that have never before performed any functions.

In *symptomatology* both *Fleischmann* and *Spiegelberg* have contributed valuable material, the former in connection with rachitis of the jaws and of the bones of the face, and the latter in describing the symptoms of fœtal rachitis. *Fleischmann* describes two changes that take place in the lower jaw; a conversion of its arched shape into a polygonal, and a change of direction in the alveolar process so that it points inward. By means of this the bone is shortened from before backwards. The change starts in the region of the canine teeth. In the upper jaw the change takes place in the neighborhood of the zygomatic arch; it does not occur so early as in the lower jaw and produces a lengthening from before backwards. In older children the alveolar process, together with the arrangement of the teeth, is changed. The molars diverge, but not as much as the upper incisors, the lateral incisors are no longer frontal, but have a lateral position, and when the jaws are closed these appear in front and to the outside of the opposite teeth in the lower jaw. The upper molars rest with their edges upon the grinding surfaces of the lower, sometimes even upon the lower edges of the latter, producing, in all, the opposite of the condition found in senile jaws. These deformities *Fleischmann* explains in the following manner: in the lower jaw by the traction upon the bone caused by contractions of the mylo-hyoid and masseter muscles; by the want of deposit of bony material in the anterior part of the jawbone; by the pressure of the lip and perhaps by the contraction of the genioglossus muscle. In the superior maxilla the deformity is explained by the pressure of the zygomatic arch preventing the outward growth of the alveolar processes, while the anterior part continues to develop. Because the changes in the arches of the jaw take place before the teeth come through: this form is of great importance to the physician, he being enabled to diagnosticate the general condition simply by the appearance of the jaws. The effects of rachitis upon the teeth are many. When rachitis develops before the sixth month of life, the incisors may not appear before the end of the first year or later; when it develops after the sixth month, the incisors appear at the proper time, but the effects are shown upon the teeth following. In children in which rickets develops before the eruption of the molars the following is observed: the incisors do not appear in their proper order; teething ceases or the first molar appears and then follows a long pause or the child reaches its twelfth month without a molar. The intervals between the appearance of teeth are longer in the rachitic than in healthy infants, in the rachitic this may extend to twenty months. *Fleischmann* refers to a case in which a child of twenty-two months had only four teeth.

Spiegelberg describes the appearance of fœtal rachitis as follows: "The fœtus is characterized by its plumpness, large body with protruding abdomen, large, sometimes hydrocephalic head, thick, short, curved extremities. The skin is thick and the adipose layer well developed, loose so that the body seems to be inclosed within a sack that is too large for it. The abdominal organs, especially the liver, are large. The changes in the skeleton are nearly the same as those of post-fœtal rachitis; the diaphyses of the extremities thickened, short, bent, frequently broken; the epiphyses swollen, soft, and cartilaginous. The ribs are in a similar condition, fractures are either not united or united by masses of callus. The disturbance in ossification is principally shown in the sternum, while the clavicle, of all bones, seems to possess greatest immunity. Chicken-breast is absent, because it is always produced by inspiration, where found it must be assumed that the child has lived for some time (*Virchow*). In the pelvis we find the flattening so characteristic of rachitis—the extension of the sacrum, the forward and downward dislocation of the promontory, the flattening of the sacral vertebræ, the widening and flattening of the ilia, the increased size of the pubic arch, all distinctly marked. The characteristic form of the entrance to the pelvis is shown, but to a much greater extent than is ever indicated in the fœtal pelvis. All of these changes are to be attributed to the traction of muscles and ligaments, as well as to the partial stoppage in the development of bone; we cannot conceive of any action of the trunk in utero, on account of its weight. It is evident that the traction may lead to infraction; the angular deformity found in the venter of the ilium must be considered an infraction. On the other hand, we can also explain why the pelvis is sometimes changed little or not at all, as the muscles may be slightly developed and therefore produce little effect. Ossification of the bones of the skull has been observed in its various stages, from a membranous bag with islands of bone to excessive thickness."

Although, theoretically, the

TREATMENT

of rachitis is perfectly simple and the indications are well marked, yet practically little has been done toward its advancement. Specifics are no longer accepted, both cod-liver oil and the phosphates have been found in many cases to be without results and have therefore been abandoned by many. The objections to cod-liver oil are: that it is not borne by children under eight months of age (*Rehn*), that even larger ones cannot take it for any length of time, and finally, when borne, that it has no effect upon the rachitic process (*Fleischmann*). Yet it is still recommended by many authors. The phosphates are universally recommended, either in the form of phosphate of calcium dissolved in hydrochloric acid, the lacto-phosphate of lime (lactophosphate de chaux of the French authorities), or in the form of the aqua calcis of the pharmacopœia (*Senator*). Phosphorus has lately been tried in the form of ol. phosphoratum (*Senator*), and *Gies* (*ib.*) recommends arsenic in small doses. *Fleischmann* speaks highly of the administration of fluorine compounds in order to increase the hardness of the enamel of the teeth. According to him it must be given between the tenth and eighteenth months, and is best given in the form of Dr. Ehrhardt's or Hunter's tooth-pastilles. These contain the "neutral" fluoride of potassium, are very agreeable to the taste, and can be easily taken. One of these a day is to be given for several weeks. Great progress has been made in the dietetics of infancy, and this naturally has its effects upon the dietetics of rachitic children. It is possible to refer to one point only in this connection. *Bernard* (of Montbrun) lives in a district where women nurse children as long as possible; when they have no children to nurse they stimulate the flow of milk by nursing puppies. *Bernard* found that in every case these puppies became rachitic, but when they were returned to their natural source of nutrition, not only did rachitis cease, but their deformities also disappeared. As a result of these observations, he treated human rachitis by allowing the infants to be nursed by dogs, and with the effect of curing the disease. It is claimed that dog's milk is much richer in salt than human milk, and that to this is due its curative properties.

Rehn's treatment of laryngospasmus must also be referred to here. When hydrocephalic complications are absent, he gives narcotics, employing either opium, chloral, or morphia in large doses. Opium he gives in doses of 0.005–0.01, repeated every three to five hours, in combination with lactate of iron; morphia 0.01 to 40.0, of which solution he gives one teaspoonful at the same times. Hydrate of chloral is given by the rectum, 0.5–1.0 to 30.0 of menstruum in the evening, because of the greater frequency of the attacks during the night.

Against the deformities that are left after rickets, orthopædic measures are employed, bandages, splints, and apparatus of various descriptions. When these are not successful, the bones may be fractured and then treated, or better osteotomy, if possible subperiosteal, with Lister's antiseptic dressing, is employed with good success.

SMALL-POX.

BY

FRANK P. FOSTER, M.D.,

OF NEW YORK,

N FOR DISEASES OF WOMEN TO THE OUT-PATIENT DEPARTMENT OF THE NEW YORK HOSPITAL.

SMALL-POX AND VACCINATION.

In the second German edition of *v. Ziemssen's* Encyclopædia, published in 1877, the author gives the following additional

BIBLIOGRAPHY.

Elsässer, J. A.: Beschreibung der Menschenpockenseuche, welche in den Jahren 1814, 1815, 1816, und 1817 im Königr. Württemb. geherrscht hat. Stuttgart, 1820.—Krause, C. F. Th.: Ueber das Alter der Menschenpocken und anderer exanthematischen Krankheiten. Hannover, 1825.—Robert: Précis historiques de l'épidémie qui règne à Marseille, et vues nouvelles sur la vaccine. Marseille, 1828.-Heim: Historisch-kritische Darstellung der Pockenseuchen im Königr. Württemb. Stuttgart, 1838.—Quincke: Charitéannalen. Berlin, 1855.—d'Espine, Marc: Etude sur la variole, la vaccine, et les revaccinations. Arch. gén. de méd., June, July, 1859.—Fiedler, A.: Statistische Mittheilungen und aphoristische Bemerkungen über die Pockenepidemie zu Dresden i. d. Jahren 1870 u. '71 u. s. w. Jahresb. d. Ges. für Natur- und Heilk. in Dresden, 1872.—Guttstadt, A.: Die Pockenepidemie in Preussen, insbesondere in Berlin, 1870–72. Zeitschr. des königl. preuss. statist. Bureaus, 13 Jahrg., 1873.—Müller, E. H.: Die Pockenepidemie zu Berlin im Jahre 1871. Viertelj. für gerichtl. Med. u. öffentl. Sanitätswesen, Bd. 17, 1872.—v. Pastau: Statistischer Bericht über das städt. Krankenhospit. zu Allerheiligen in Breslau für das Jahr 1871, nebst einem Anhang, enthaltend Beiträge zur Pockenstatistik nach den Erfahrungen aus der Pockenepidemie 1871–72. Breslau, 1873.—Meyer, Lothar: Ueber Pocken beim weiblichen Geschlecht, Beitr. zur Geburtsh. und Gynäk. Berlin, 1873.—Meyer, L.: Bericht aus d. städt. Pockenlazareth. Deutsche Klinik, 1870, Nos. 6–10.—Petters, W.: Die Blatternepidemie in Prag. Prager Viertelj., 1876, Bd. I. und II.—Erismann: Zur Anatomie der Variola hæmorrhagica. Sitzungsb. d. mathem. naturwissenschaftl. Klasse der kaiserl. Akademie der Wissensch. Wien, 1868, Bd. 68, Abth. II.—Wyss, O.: Zur Anatomie der hämorrhag. Pocke und der Purpura variolosa. Arch. f. Dermat. u. Syph., Bd. III., 1871.—Desnos: Considération sur le diagnostic, le prognostic et la thérapeutique de quelquesunes des principales formes de la variole. L'Un. méd., 1870, No. 73.—Huchard: Etude sur les causes de la mort dans la variole. Arch. gén., 1871.—Quinquaud: Note pour servir à l'histoire des lésions viscérales diffuses de la variole. Gaz. des hôp., 1870, No. 97.—Weigert, C.: Anatomische Beitr. zur Lehre von den Pocken. Heft I.: Pockeneffloresc. der äusseren Haut. Breslau, 1874.—*Ibid.*, II. Theil: Ueber pockenähnliche Gebilde in parenchymatösen Organen und deren Beziehung zu Bakteriencolonien. Breslau, 1875.—Golgi: Sulla alterazioni del midollo della ossa nel vajuolo. Revista Clinica, 1873.—Adler, H.: Die während und nach der Variola entstehenden Augenkrankheiten. Viertelj. f. Dermat. u. Syph., 1874.—Brouardel, P.: Etudes sur la variole. Lésions vasculaires (cœur et aorte). Arch. gén. de méd., Dec., 1874.—Henle: Pathologische Untersuchungen. Berlin, 1840.—Keber: Ueber die mikroskopischen Bestandtheile der Pockenlymphe. Virch. Arch., 1868, Bd. 42.—Salisbury: Microscopic Examinations of Blood and Vegetations found in Variola, etc. New York, 1868.—Burdon-San-

derson: Introductory Report on the Intimate Pathology of Contagion. Twelfth Report of the Medical Officer of the Privy Council, with appendix, 1869. Lond., 1870.—Chauveau: Nature du virus vaccin. Détermination expérimentale des éléments qui constituent le principe actif de la sérosité vaccinale virulente. Comptes rendus hebdomadaires des séances de l'Académie des Sciences, 1868, T. 66, p. 289. Further articles by this author upon the same subject: *Ibid.*, T. 66, pp. 317, 359; T. 67, pp. 696, 898, 941; T. 68, p. 828.—Reiter: Beiträge zur richtigen Beurtheilung u. s. w. München, 1846.—Kussmaul: Zwanzig Briefe über Menschenpocken- und Kuhpockenimpfung. Freiburg, 1870.—Friedberg: Menschenblattern und Schutzpockenimpfung. Breslau, 1874.—Bohn: Handbuch der Vaccination. Leipzig, 1875 (a carefully prepared collation of the literature of the subject).—Eulenberg: Ueber die nothwendige Zahl der Pusteln bei der Vaccination und Revaccination. Vierteljahrschr. f. ger. Med. u. öffentl. Sanitätswesen, Bd. 18, 1873.—Burchard: (on the same topic) Militärärztl. Zeitung, 1872, p. 536. —Pissin: Die beste Methode der Schutzpockenimpfung. Berlin, 1874.—Chauveau: De l'autonomie de la vaccine. Annales de dermatol. et de syphil., 1870, No. 5.—Müller: Ueber Pockenimpfung und über die Bedeutung der Glycerinlymphe für die öffentliche Gesundheitspflege. Vierteljahrschr. f. gerichtl. und öffentl. Med., 1869, Bd. II.

Vaccinal Syphilis.

Fassani: Gaz. medic. di Milano, 1843.—Wegeler: Preuss. Vereinszeitung, 1850, No. 11.—Pacchiotti: Sifilide transmessa per mezzo della vaccinazione in Rivalta, presso Acqui. Torino, 1862.—Viennois: Arch. gén. de méd., 1860, Vol. I., p. 642; Vol. II., pp. 32, 297.—Pellizari: Gaz. méd. de Lyon, 1862, No. 10. —Auspitz: Die Lehre vom syphilit. Contagium. Wien, 1866.—Rahmer: Diss. Breslau, 1869 (Köbner).—Köbner: Arch. f. Dermat. und Syph., 1871, Jahrg. III.—Hutchinson: Med.-Chir. Trans., Vol. 54, 1871.—*Idem*, Lancet, Apr. 7th, 1873.—The remaining bibliography of vaccinal syphilis may be found in the works cited.

For the history of small-pox consult the well-known works of Curt Sprengel, Hecker, Häser, and August Hirsch.

The present writer would add the following:

Lüders: Versuch einer kritischen Geschichte der bei Vaccinirten beobachteten Menschenblattern, etc. Altona, 1824.—Gayton, W.: Notes on Small-pox and its Treatment. London, 1873.—Grieve, R.: The Influence of Vaccination, Age, Sex, and Occupation on the Mortality in Small-pox. London, 1872.—Hamel: Du rash variolique. Thèse, Paris, 1870.—Colin, L.: La variole au point de vue épidémiologique et prophylactique. Paris, 1873.—Chauveau: Théorie de la contagion médiate ou miasmatique, encore appelée infection: les voies de l'infection sont-elles spéciales aux agents virulents des maladies dites infectieuses? Comptes rendus des séances de l'Acad. des Sciences, T. LXVII., No. 19, p. 941.—Welch, W. M.: Report of the Board of Health of the City and Port of Philadelphia, to the Mayor, for the Year 1872. Philadelphia, 1873.—Welch, W. M.: Small-pox in the Pregnant Woman and in the Fœtus. Philadelphia Med. Times, May 25th, 1878. — Farquharson: Case of Small-pox . . . believed to have Originated from the Contagion of Chicken-pox. Lancet, 1877, Vol. I., p. 879.—Rigden, G.: Influence of Maternal Small-pox on the Fœtus. Brit. Med. Journ., 1877, Vol. I., p. 229.—Marson, J. F.: On Small-pox and Vaccination, etc. Med.-Chir. Trans., Vol. XXXVI., 1853.—Bryce, J.: Practical Observations on the Inoculation of Cow-pox, etc. Edinburgh, 1809.—Ceely, R.: Observations on the Variolæ Vaccinæ, etc. Worcester, 1840.—*Idem:* Further Observations, etc. Trans. Prov. Med. and Surg. Asso.—Hering, E.: Ueber Kuhpocken an Kühen. Stuttgart, 1839.—Bousquet, J. B.: Sur le cow-pox (petite-vérole des vaches) découvert à Passy (près Paris) le 22 Mars, 1836. Paris, 1836.—Depaul: Expériences . . .

avec le cow-pox ou vaccin animal, etc. Paris, 1867. [Gives the history of the Beaugency case of cow-pox.]—*Idem:* Nouvelles recherches sur la véritable origine du virus vaccin. Paris, 1863.—*Idem:* De l'origine réelle du virus vaccin. Paris, 1864.—*Idem:* Sur la vaccination animale. Paris, 1867.—BOUVIER: De la vaccine du cheval. Bull. de l'Acad. Imp. de Méd., 1863–4, T. XXIX., p. 390.—DENARP-DECANTELEU: Monographie des cicatrices de la vaccine, etc. Paris, 1851.—CHAUVEAU, VIENNOIS, MEYNET: Vaccine et variole; nouvelle étude expérimentale sur la question de l'identité de ces deux affections. Paris, 1865.—BOUSQUET: Nouveau traité de la vaccine et des éruptions varioleuses. Paris, 1848.—STEINBRENNER: Traité sur la vaccine, etc. Paris, 1846.—SEATON: A Handbook of Vaccination. London, 1868.—BALLARD: On Vaccination; its Value and alleged Dangers. London, 1868.—CORNIL: Anatomie de la pustule de la variole et de la vésicule de la varicelle. Jour. de l'Anat., Mar. 1st, 1866. —HAMERNÍK: Contagium, Epidemie und Vaccination. Prag, 1867.—DEPAUL: La syphilis vaccinale. Paris, 1865. — AUZIAS-TURENNE: Les virus au tribunal, etc. Paris, 1868.—JONES: Researches upon "Spurious Vaccination," or the Abnormal Phenomena accompanying and following Vaccination in the Confederate Army during the recent American Civil War, 1861–65. Nashville, 1867. —HART: The Truth about Vaccination. London, 1880.—WEIR: Animal Vaccination (also editorial remarks). Lancet, 1878, II., p. 29.—GREENE: Animal Vaccination. Brit. Med. Jour., 1878, II., p. 889.—VON RINECKER: Ueber Vaccinations-Syphilis. Viertelj. f. Derm. u. Syph., V., 1878, p. 259.—MARTIN: On True Animal Vaccination. Med. Record, XIII., 1878, p. 304.—KLEBS *et al.:* Debatte über Kuhpockenimpfung im Vereine deutscher Aerzte in Prag. Viertelj. f. Derm. u. Syph., IV., 1877, p. 214.—(Editorial): The Pathology of Vaccinia. Lancet, 1878, II., p. 342.—BRAIDWOOD: The Comparative Merits of Animal Vaccination and Arm-to-Arm Vaccination. Edinb. Med. Jour., Nov., 1878, p. 385.—MARTIN: Animal Vaccination and its Difficulties. Med. Times and Gaz., 1877, I., p. 403.—WARLOMONT: Animal Vaccination. Brit. Med. Jour., 1877, II., p. 183.—MARTIN: A few Words on "Unfortunate Results of Vaccination." Boston Med. and Surg. Jour., XCVI., 1877, p. 125.—WARLOMONT: Remarks on Animal Vaccination. Brit. Med. Jour., 1879, II., p. 926.—GAST: Experimentelle Beiträge zur Lehre von der Impfung. Schmidt's Jahrb., 1879, No. 8, p. 201.—(Editorial): Animal Vaccination. Med. Times and Gaz., 1879, II., p. 610.—HART: Preliminary Report on Animal Vaccination in its Relation to Proposed Legislation. Brit. Med. Jour., 1879, II., p. 843.—FLEMING: Human and Animal Variolæ; a Study in Comparative Pathology. Lancet, 1880, I.

No very notable addition to the literature of small-pox has been made since the first edition of Dr. *Kurschmann's* article was published. In our extension of the author's bibliography of vaccination, we have given some of the older works, mainly because physicians find it difficult, as a rule, to look up the literature of this subject.

The author's additions in the second German edition (1877) are substantially as follows:

SMALL-POX.

COMPLICATIONS AND SEQUELÆ.—*The Eye.*—Pustules occasionally from upon the conjunctiva of the lids, and more rarely upon that of the globe or upon the mucous membrane of the lachrymal tract. Their occurrence upon the cornea is absolutely denied by *Adler*. The cornea and the iris are not attacked before the second week of the disease.

We then find superficial and parenchymatous keratitis and, especially in severe cases of the confluent form, extensive deep suppuration of the cornea, resulting not very seldom in panophthalmia. Iritis, generally insidious in its course and not infrequently complicated with choroiditis, never occurs, according to *Adler*, before the twelfth day. Acute glaucoma is very rarely observed. Now and then retinal hemorrhages occur in hemorrhagic cases; opacities of the cornea, staphyloma, adhesions of the iris, and even closure of the pupil are common sequelæ. Ectropion and other distortions of the lids are likewise observed, and occasionally obstinate eczema of the lids or chronic catarrh of the conjunctiva. Nervous disturbances of the visual apparatus also are mentioned by *Adler*.

The ear suffers oftener than the eye. In 168 cases, *Wendt* found aural lesions in 98 per cent. Pustules were almost always found upon the external ear, more rarely extending into the cartilaginous portion of the canal. In the bony portion, the characteristic efflorescence was never found, but frequently hyperæmia and swelling, occasionally reaching to the drum-membrane. Now and then inflammatory manifestations were found upon the mucous membrane of the middle ear, but never pustules. Although changes in the naso-pharyngeal space cannot be reckoned among the complications, it should be said that, in severe cases, tumidity, with purulent infiltration of the epithelium, extends to the mouths of the Eustachian tubes; stenoses of the tubes and catarrh of the middle ear are frequently dependent upon this cause. Impairment of hearing and even complete deafness not uncommonly results from these affections. Chronic catarrh and caries may also ensue.

Ulcerations of the *nasal mucous membrane* occur, although somewhat rarely, and may end in occlusion of the nostril.

Affections of the *locomotor apparatus*, mostly limited to the joints, vary in different epidemics, and in different periods of the same epidemic. Hence the diversity of statements as to their frequency. Acute articular inflammations are the most common, with serous or sero-purulent effusion. They generally affect the capsule or the ligamentous structures, and more rarely the cartilage or even the ends of the bones. The larger joints are most apt to be involved—most commonly, according to my experience, the shoulder, and then the knee. I have generally seen only a shoulder affected, but sometimes as many as from four to six joints. The arthritis generally begins during the suppurative stage—most commonly in the discrete and the confluent forms of the disease, seldom and with less intensity in varioloid, in which latter case there is no exudation or a purely serous one. In one case of pustular hemorrhagic variola, I found a bloody and purulent collection in each knee-joint.

In children, particularly, *catarrhal pneumonia* may supervene upon the bronchitis, which almost always occurs in well-marked cases. The disposition is shown, too, even in adults, and in persons predisposed to phthisis the disease is often kindled by the inanition that follows the process of pock-formation. *Croupous* and *hypostatic pneumonias* are not uncommon, the former during any period of the eruptive stage, the latter towards the end and in cases following a protracted course. *Pleurisy* is still more common. It seldom comes on before the twelfth day, but is apt to appear then quite suddenly. The tendency to result in empyema is quite remarkable. I have never found the exudation purulent at the very first, but it generally becomes so in the course of three or four days. I have almost always been struck with the thin character of the pus. As a rule, variolous empyema is extremely rapid and malignant in its course. Although I cannot say that the treatment by incision and subsequent irrigation affords an actual prospect of cure, yet it is worth trying. My own observations, occurring before the present method came into general use, do not constitute proper data for settling this question, nor am I aware of any published by others. I believe, *à priori*, that the prospect is comparatively dismal. In two cases I have seen the course so rapid that death occurred on the fourth and sixth days, respectively, after the first symptoms were observed, quite as in the hyperacute empyema of *Traube*. In both patients the eruption was confluent. The frequency of pleuritic affections, like that of most of the graver complications, bears a certain relation to the intensity of the variolous process.

In several instances I have seen *pericarditis* in conjunction with pleurisy, and in one case without the latter. I have met with one case of ulcerative *endocarditis* in confluent small-pox.

Diphtheria, affecting the soft palate and neighboring parts, is mentioned as a common complication in almost all the severe forms of the disease. I have found it almost always present in the hemorrhagic form. It seems to me, however, that confluent pocks upon the mucous membrane may not infrequently lead to the false inference that true diphtheria is present, especially when they give rise to irregularly serrated loss of substance.

Severe *affections of the larynx*, such as ulcers of the mucous membrane, perichondritis, and necrosis of cartilage, may give rise to chronic hoarseness, or even complete aphonia.

Inflammations of the salivary glands, regarded by the older writers as of unfavorable significance, seem to have grown less common of late years. In the few instances that I have seen, they had no influence, other than a local one, upon the course of the disease.

The *abdominal organs* are not often affected. Like other recent writers, I have never seen peritonitis without a local cause. Obstinate diarrhœa occasionally occurs, and may far out-last the small-pox. *Sydenham* mentions a variola dysenterica. *Trousseau*,* who seems to have met with diarrhœa frequently, states that it always appears during the first few days of the process of pock-formation. In his experience, it disappeared by the fifth day at the latest, and never produced any ill effects.

The *genital organs in women* are particularly liable to be affected.

---

* Med. Klinik, deutsch von *Kullmann*, S. 18.

In pregnant women the greatest danger of abortion is incurred—the greater, the severer is the disease. I have never known it fail in hemorrhagic small-pox. As a rule, it is more easily produced in the latter months of gestation, although there is a somewhat marked tendency to it during the first three months. While we are not fully acquainted with the causes, no doubt the great proneness to metrorrhagia and the frequent death of the fœtus are two important elements.

In non-pregnant women, excessive, premature, or too frequent menstruation is notably apt to occur.

There is a certain liability to inflammatory affections of the *testicle* and the *ovary*. Inflammations of the latter organ and its surroundings have not, thus far, been studied very thoroughly. *Variolous orchitis*, however, has received particular attention from French authors, and *Trousseau* speaks of it as quite common. It may affect the parenchyma, the investments, or both together. I myself have seen it but four times in 432 men.

More has been said about *pyæmia* than its frequency justifies. It is most apt to occur in the suppurative and the desiccative stages of confluent cases. Its occurrence cannot, of course, be wholly denied—in fact I have seen two cases in which death could be accounted for in no other way, and in which the autopsies showed metastatic abscesses in the liver and the lungs.

Severe small-pox is almost always followed by excessive *exhaustion*, which proves tedious in its duration. I have even seen slight dropsy, which I have felt constrained to refer to the extreme anæmia. Dropsical symptoms may, of course, depend upon chronic nephritis, which, however, is, according to my experience, a very rare sequel of small-pox.

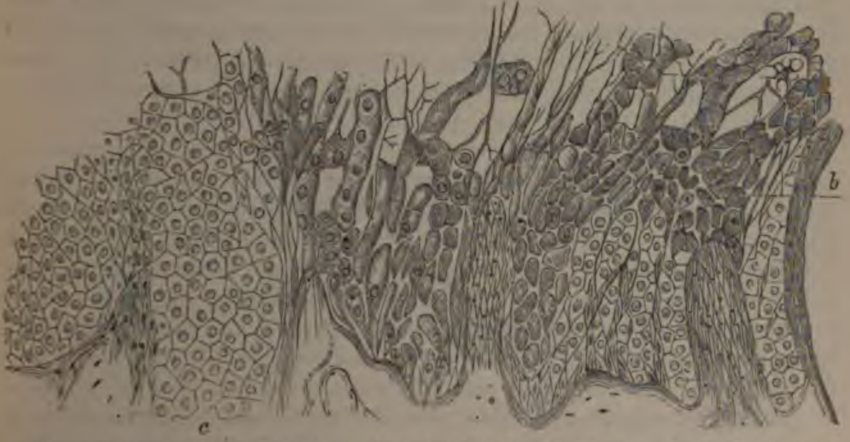
PATHOLOGICAL ANATOMY.

Our present data *do not warrant the conclusion that the micrococci are either the contagium or its vehicle.* *Wolf's* experiments, in which by the most careful filtration he failed to obtain lymph wholly free from microspheres, tend to vitiate the results of *Chauveau's* experiments with filtered lymph. The whole subject of the relations of the micrococci and the disease needs to be more thoroughly investigated before any conclusions can be drawn.

The first step in the *formation of the pock*, the red spot, is due to a circumscribed hyperæmia of the papillary body, extending, according to *Bärensprung*, through the whole thickness of the cutis. The succeeding papule is caused by a peculiar process in the epidermis, and is thus distinguished from many other papules arising from circumscribed swellings of the cutis.

Weigert has recently shown that the "cloudy swelling" of the cells of the rete Malpighii, formerly supposed to be due to inflammation, is of a peculiar, necrobiotic nature, of which the surrounding inflammation is a result. Sections of the papule, in its formative stage, show at their centres striking changes of these cells, which are found to have been transformed into cloudy flakes, more or less opaque, very irregular in their shape and size, and, what is specially significant, destitute of nuclei. These elements are sometimes smaller, sometimes much larger than the normal cells of the rete; they may be roundish, or furnished with angles and projections, or perforated, or bandlike, in the most varying arrangement. These changes are most marked in the interior of the mass, whilst toward the periphery we may find traces of nuclei along with flakes destitute of nuclei. There may be a sharp line of demarcation from the healthy parts, or the transition may be gradual. The chief collections of these elements, which may be surrounded by lesser ones (especially if the lesion is a large one), are seated for the most part in the neighborhood of a hair-follicle or of the mouth of a sweat-gland—a relation which quite conforms to the well-known predilection of the rash for those structures.

Weigert believes, and on good grounds, we think, that this flaky degeneration of the cells of the rete, always to be found in the nascent variolous papule, must be ascribed to a sort of necrobiosis, constituting the first specific effect of the small-pox poison upon the epidermis; and he assumes that the flakes, like any other dead material, irritate the surrounding tissue, provoking, as a secondary matter, the inflammatory phenomena of the rash. He calls the degeneration "diphtheroid," from its resemblance to the epithelial changes found by *Wagner* in diphtheria.



From a young pock (after *Weigert*). *a.* A principal diphtheroid collection. *b.* An adjoining one. Both show the peculiar flaky degeneration of the epithelia. *c.* Excretory duct of a sweat-gland, near which (upon the right) are cloudy and swollen epithelia, with their nuclei still discernible.

It has thus far been assumed that the papule is at first a solid swelling of the epidermis, but *Weigert* has shown that very small spaces are to be found in the rete Malpighii while yet the efflorescence is scarcely at all prominent. As the effusion increases, the cells, more or less changed, are separated, and the overlying horny layer is raised bodily, so that a little vesicle is formed upon the summit of the papule. The pressure of the effusion not only separates the changed epidermal elements, but masses them in groups, and compresses them into membranous and string-like formations constituting the meshes of the pock. For this important point in the doctrine of pock-formation we are indebted to *Auspitz* and *Basch.*[*] *Bärensprung,* however, still denies the partitioned structure of all but confluent pocks.

With the growth of the pock, the rete-cells immediately adjoining the meshwork undergo marked proliferation, forming a sort of wall at the periphery, of no little significance in explaining the umbilication, as we shall presently see. The fluid is clear only at the very first, being speedily rendered turbid by necrotic matter, little clots of fibrine, and a few pus corpuscles, which latter have evidently migrated from the vessels of the cellular tissue, and are not, as was thought until lately, formed from the epithelium itself. As the pock grows and matures, they force themselves into the loculi in such numbers that they sometimes wellnigh cover the trabeculæ. *Weigert,* whose exposition we have mainly followed, considers as well the cell-proliferation as the transudation, and the suppuration as secondary irritative effects of the diphtheroid cell-masses.

The vexed question of the mechanism of *umbilication* is very simply explained by *Auspitz* and *Basch* as due to a very rapid swelling of the peripheral cells, so that their increase in bulk outstrips that of the elements at the centre of the pock. Other authors, however, including *Weigert,* concede to the central meshwork a share in the process, and this is certainly true of some pocks. It has been supposed that the trabeculæ acted as bridles, binding the dome of the pock to the floor, and *Rindfleisch* attributes the same action to the excretory duct of a sweat-gland.

As the pustule fully matures, the dome generally becomes smoothed out, either by the central retinaculum becoming more yielding or by the trabeculæ being torn by the increasing pressure of the fluid contents of the pock. When desiccation begins, a depression often forms again (even in pocks that before had none), because the centre sinks on account of the evaporation of the fluid contained in it, whilst the peripheral wall is still maintained by epithelial growth. *Auspitz* and *Basch* have called this the "secondary umbilication," or "umbilication of desiccation."

Early in the progress of the pock, the papillary layer underlying its centre is swollen, but later on I have generally found it somewhat flattened out again, and to this variation I attribute the divergent views of *Auspitz* and *Basch* and of *Weigert* in regard to the behavior of the cutis. This sort of pressure-atrophy often persists after the crust has fallen, and thus gives rise to more or less enduring shallow depressions, which, however, should not be confounded with the ugly, radiated scars that are so often left, the latter being caused in quite a different way. Especially in confluent small-pox, often also in the discrete form, and occasionally even in varioloid, the papillary layer beneath the pustule is so decidedly involved in the inflammation that it actually ulcerates; and the pocks (generally with their chambers much enlarged by reason of the destruction of the more delicate partitions) then contain, along with pus-corpuscles, shreds of the papillæ. More or less of the latter structure, however, generally escapes destruction, and thus it happens that the crusts remain adherent for a long time, dipping down as they do into the spaces between these undestroyed portions. Upon the extent and form of this destructive process depend the size and shape of the radiated scars.

---

[*] Virchow's Archiv, Bd. 28.

The process of incrustation begins at the central and superficial portion of the pock, and advances to the deep portion and toward the periphery. The crust is made up of the elements of the framework and of the solid constituents of the contents—detritus and especially dried pus-corpuscles. Upon most parts of the body, especially in the discrete form, it is closely connected and more or less continuous with the sound epidermis, being made up of the remains of the former dome of the pock. The newly formed epidermis advances centripetally beneath the crust, so that the latter becomes encapsulated, as it were, between the old epidermis and the new. This relation is best observed on the palms and soles, where the outer layer of the capsule is thick.

The anatomy of the skin lesions has not been very thoroughly studied in the various forms of small-pox, but we may assume that it does not differ essentially from the type. The warty nodules of variola verrucosa are due to a primary marked growth of the papillæ.

There is scarcely anything special about *hemorrhagic pocks*. *Wagner** and *Wyss* † have thoroughly refuted *Erismann's* idea ‡ that there is a particular tendency to hemorrhage into the hair-sacs. The contents of the pocks are simply bloody, instead of purulent or serous. This is almost always the only peculiarity. Besides hemorrhagic pustules, we generally find diffuse hemorrhages into the substance of the cutis; from little petechiæ up to large patches. In the less severe cases, the hemorrhage occasionally spares the papillary layer, but in the graver forms it affects all the layers of the cutis and even the subcutaneous fatty tissue —the latter especially in purpura variolosa, in which the patient dies before a single pustule has formed. In this form, too, we see the most extensive hemorrhages, so that the whole skin, particularly upon the abdomen, is continuously invaded, as well as the panniculus adiposus. *Wagner* has shown the probability that the hemorrhage does not take place by the rupture of small vessels, but by a diapedesis of the blood-corpuscles through their unbroken wall. In spite of many recent attempts, however, there has been no positive recognition of changes in the blood-vessels, explanatory of this occurrence.

---

* Die epithelialen Blutungen. Arch. der Heilkunde, IX.

† Arch. f. Dermatol. u. Syphil., III., pp. 529 et seq.

‡ Sitzungsber. der Wien. Acad., 1868.

As regards the *condition of the internal organs*, we shall see that the purely pustulo-purulent [pustulös-eiterige] form of variola vera differs materially from well-marked purpura variolosa, as *Ponfick*, in particular, has quite recently maintained; whilst the various forms of pustular hemorrhagic small-pox correspond more closely with variola vera in their lesions, or else show a transition from the latter to purpura.

Variola vera shows itself upon the *mucous membranes* either (*a*) specifically, in the form of pustules or of profuse purulent infiltration of the epithelial (middle) layer; or (*b*) non-specifically, in the shape of catarrhal, croupous, and diphtheritic processes. Practically, the histology of pocks of the mucous membrane has not been studied at all as yet. The intensity and extent of their development are for the most part in direct ratio with the changes in the skin. They not infrequently precede the latter, red spots and papules being found during the initial stage.

Those mucous membranes that are exposed to the air are preferably affected. Accordingly, we find the rash the thickest and the deepest in the *respiratory tracts*. It is pretty constantly met with in the nose, in the mouth, and upon the tongue. The epithelium of the latter, indeed, is not seldom lost over large patches. The tonsils are favorite seats of the manifestations, as well as the soft palate and the whole naso-pharyngeal space, in which latter they reach to the openings of the Eustachian tubes, and even extend into the tubes, in the form of swelling and purulent infiltration of the epithelium.*

Pustules are often observed in great numbers in the *trachea*, as far down as the bifurcation, where they are apt to be particularly abundant and occasionally confluent. According to *Wagner*, they may be traced into the bronchi as far as those of the second and third orders. The mucous membrane is diffusely swollen and bluish-red, and is often covered with great patches of a dirty grayish-white coat. The smaller and the very finest bronchi show nothing but a more or less intense catarrh. The bronchial tubes as a whole usually contain a yellowish, bloody, occasionally very thin mucus. In connection with the bronchitis, catarrhal pneumonia is quite common, and there is a manifest tendency to lead to phthisis in those disposed to it.

In the *digestive tract*, pustules are limited to the œsophagus, occurring most abundantly or exclusively in its upper parts. Only catarrh and slight hemorrhages are found in the *stomach* and *intestine*, ulceration of the follicles, however, being now and then met with in the latter, with secondary swelling of the mesenteric glands. It is more than doubtful if pustulation really occurs in the stomach and intestine. It has occasionally been observed in the very lowest part of the rectum, close to the anus. Pustules and diphtheritic processes are found upon the *vulva* and in the *vagina*, but pustules are never met with in the *bladder*, and in the *urethra*, always discrete, only close to the meatus.

---

* *Wendt, l. c.* We are especially indebted to this author for data in regard to the state of the ear in small-pox, founded upon the examination of 168 cases. The striking fact was revealed that morbid changes (limited to those due to the variolous process) were present in 98 per cent. There were almost always pustules in the pinna, and less often at the beginning of the cartilaginous portion, whilst the few instances of affections of the bony portion and upon the membrana tympani showed only hyperæmia and swelling. These latter changes were found now and then even in the middle ear, where pustular affections never occur.

True *pocks upon serous membranes* are but the fables of a bygone age. Hyperæmia, inflammation, and hemorrhage are, however, extraordinarily common. The *pleura* is the favorite seat of these lesions, while it is still doubtful if *peritonitis* occurs as the result of the small-pox poison.

The changes in the *great abdominal glands*—the *liver*, the *kidneys*, and the *spleen*—are very important. Authors are at variance in regard to them. We sometimes find cloudy swelling of the parenchyma of the liver and of the cortical substance of the kidney, and sometimes an undoubted acute fatty degeneration of these parts—so intense and so widespread as to remind one of phosphorus poisoning. In many cases these organs are found quite normal, especially the last-named, in pustular forms of the disease; and these cases include, I believe, those in which death has occurred early, before the essential changes in the cells have become developed, and those in which, where death has taken place at a late period, the organs have recovered from the cloudy swelling and have escaped the fatty degeneration. The latter change is the sequence of the former—being very sudden or gradual in its development, according to the intensity, etc., of the disease. The *bile* is almost always strikingly pale and thin in pustular small-pox. In the hemorrhagic pustular form, I have quite generally found cloudy swelling and fatty degeneration of the organs mentioned. Their absence, however, according to *Ponfick* and to my own observations, is rather characteristic of purpura variolosa. In these cases the liver is generally of the normal size and of a dusky color, but markedly firm and hard.

In the early stages of variola vera the spleen is almost always manifestly swollen, and its substance is soft and pale-red; whilst in the later stages it is quite commonly found unchanged. In purpura, on the other hand, we always find it small, remarkably firm, and showing on section a dirty dull-red and glistening surface, occasionally, according to *Ponfick*, with markedly enlarged, pure white or yellowish-white follicles. In 24 autopsies I have found this condition almost constant, and *Golgi*,* in 25 examinations of persons dead of hemorrhagic small-pox, always found the spleen small and hard.

The attempt to establish a more complete distinction between variola vera and purpura variolosa, on the strength of the widely different conditions of the great abdominal glands in the two forms, seems to us ill founded. In the post-mortem examination of a case of purpura, we have before us *an entirely different stage of the disease* (*the initial stage*), *impressed in its nature by a hemorrhagic process which we do not under-*

---

* *C. Golgi.* Sulle alterazioni del midollo delle ossa nel vajuolo. Rivista Clinica, 1873; Med. Centralbl., 1874, No. 7.

stand. Let a case of simple variola vera come to autopsy in the initial stage, and there too we should not yet find the organs in the state that they assume in the more advanced disease.

Weigert has made the very remarkable observation that, especially in the early stages of pustular cases (but he has also found it twice in hemorrhagic small-pox), sharply defined collections of bacteria, apparently within the blood-vessels, are found in the liver, the spleen, the kidney, and the lymphatic glands. In some cases the surrounding parenchyma was quite unchanged; in others the neighboring cells showed a peculiar degeneration, appearing as irregular flakes and heaps, destitute of nuclei, of a singularly faded color, and of a faint lustre, between which lay single free nuclei. At the periphery of the little collections the form of the original cells could still be plainly recognized, but they were opaque and destitute of nuclei. Between them were bodies resembling pus-corpuscles. In many of the immature collections the form of the cells was perfectly preserved, but they were without nuclei and of a faded appearance. *Weigert* correctly emphasizes the great resemblance of these changes to the diphtheroid processes upon the skin, and speaks of *"pock-like formations" in parenchymatous organs.* He attributes the cell-degeneration to the direct action of the bacteria. In a few cases in which he failed to find the microspheres in the centre of the collection, he assumed that they were changed past recognition or had disappeared altogether.

In cases where there is marked fatty degeneration of the liver and kidney, the *muscular tissue of the heart* is also flabby, yellowish, and soft, whilst in purpura, according to *Ponfick*, it is strongly contracted, firm and brownish-red. *Brouardel* has recently sought to show that inflammations of the endocardium and of the intima of the aorta are of very frequent occurrence in the severe forms of small-pox. These affections would seem to resemble those found in the same structures in pyæmia, and to differ decidedly from those of acute articular rheumatism, in that they show preference for the valves.* German authors scarcely mention affections of the heart and blood-vessels as observed in recent epidemics. I will simply mention again my observation of ulcerative endocarditis.

Hyperæmia or œdema of the *brain* is occasionally found.

In the hemorrhagic forms of the disease, besides the lesions already mentioned, *extravasations of blood, of greater or less extent, are found in almost all the internal organs.* As upon the skin, so upon the serous membranes, we find numerous petechiæ and extensive ecchymoses, and occasionally bloody exudations into the serous sacs, especially the pleura and the pericardium. In the loose cellular tissue of the mediastina and of the true pelvis, in the retro-peritoneal connective tissue, and in the capsule of the kidney, the hemorrhages are still more abundant and more diffused than within the serous membranes. Many are disposed to associate the hemorrhages in the latter region, often enormous, with the pain in the back which is so especially severe in the hemorrhagic form. *Hemorrhages occur, too, from almost all the mucous membranes.* In the air-passages we see them from the nose down to the trachea and the bronchi. As regards the digestive tract, they are most abundant in the pharynx, the œsophagus, the stomach, the colon, and the rectum; whilst, according to *Ponfick*, they are far rarer and less copious in the ileum.

---

* Etudes sur la variole. Lésions vasculaires (cœur et aorte). Arch. Gén. de méd., Dec., 1874.

In the *uro-genital system* hemorrhages take place from the mucous membrane of the uterus and of the Fallopian tubes, and occasionally into the parenchyma of the testes. The substance of the kidney almost always escapes, whilst its pelvis, the calices, and the ureters are quite constantly the seat of characteristic hemorrhages.*

Like the kidneys, the other parenchymatous organs, the liver, the spleen, and the central nervous system, are very seldom the seat of hemorrhage. Infarctions of the lungs are very commonly found, but they are generally of very moderate size. According to *Golgi* (l. c.), notable hemorrhages are quite constant in the *marrow of the bones.* The marrow is then of a deep, dusky-red color, and almost entirely fluid, like blood. Together with a marked increase of the red blood-corpuscles, the microscope shows a very material diminution of the white medullary cells. In the suppurative stage of pustular variola, according to *Golgi*, the marrow is of a pap-like consistence and of a grayish-red color, and shows a very noticeable increase of the giant-cells, and (although to a less extent) of the white medullary cells.

TREATMENT AND PROPHYLAXIS.

Every large town should have its small-pox hospital—not in the thickly peopled part, but on the outskirts (near enough, however, to spare the patient a long journey). As regards its plan, the barrack system is most to be recommended, for it gives the largest, airiest, and best ventilated rooms, with the least possible material. In no other disease is so great an amount of air-space needed. Under no circumstances would I consent to less than 1500 cubic feet for each bed, and there ought to be much more. Special rooms should be set apart for patients whose disease is yet of doubtful nature, and who are to be kept under observation. Every small-pox patient, no matter what may be his social rank, should be compelled to go to the hospital, unless he can be thoroughly isolated at home. The hospital should have associated with it a disinfection establishment of proportionate size and well conducted.

The contagium is extraordinarily persistent and difficult to destroy. Chlorine, sulphur, and especially a high degree of heat are most destructive to it. The patient's clothing and bedding should be exposed to the air for weeks together before being used again. The room should not be used until it has been specially disinfected, preferably with chlorine. Great care should be taken to avoid infection from dead bodies.

---

* See *Unruh's* full account of these phenomena, in Archiv der Heilk., XIII., pp. 289 et seq.

VACCINATION.

The enormous advantages of humanized lymph lie in the facts *that it may be had in any desired amount, that it is more easily preserved, and that it is more certain to produce an effect.*[*]

Müller usually employs lymph mixed with two parts of glycerine and two of distilled water. This mixture may be further diluted with eight times its bulk of water without materially impairing its efficiency. *Reiter*[†] found a slight effect even from lymph mixed with two hundred parts of distilled water. In taking lymph, the admixture of blood need be avoided only in case the vaccinifer is syphilitic. *Reiter* has even shown that vaccination may be successful with the blood alone, which shows one-twelfth the efficiency of that of lymph.

---

[*] The present writer would suggest that these statements do not rest upon ascertained facts.

[†] Bayr. ärztl. Intell.-Bl., 1872, Nr. 15.

MALARIAL DISEASES.—DENGUE.

BY

HENRY M. LYMAN, M.D.,

OF CHICAGO,

PROFESSOR OF PHYSIOLOGY AND OF DISEASES OF THE NERVOUS SYSTEM IN RUSH MEDICAL COLLEGE,
CHICAGO, ILL.

MALARIAL DISEASES.

BIBLIOGRAPHY.

Oeffinger: Blätter für Heilwissenschaft, München, 1873, Jg. IV., No. 14.—Burdel: Bull. gén. sc. Thérap., 1873, pp. 409 u. 529.—Gimbert: Arch. géner. de méd., 1873, pp. 129 u. 709.—Bourel-Roncierc: Les Lymphangites Primitives de Rio-de-Janeiro. Arch. méd. Navale, tome VIII., 1873, p. 335.—Castan: Montpelier méd., 1873, Juin, p. 495.—Bleiweiss: Memorabilien, 1873, Heft 3, S. 109.—Dorsch: Deutsche Klinik, 1874, No. 11.—Mahé: Arch. de méd. Navale, 1874, Fev., p. 74, Mars, p. 142, Mai, p. 289, Juin, p. 354.—Masse: Rec. de même de méd. Milit., 1874, Sep., Oct., p. 417.—Colin: L'Union méd., 1874, No. 4.—Fuhrmann: Deutsche militär. Zeitschrift, 1874, No. 12, S. 635.—Kafka: Wien. med. Presse, 1874, No. 35, S. 821.—Petermann: Zur Wirkung der Eucal. glob. Dissert. Greifswald, 1874.—Hirsch: Berlin. klinische Wochenschr., 1874, No. 30, S. 364.—Arnstein: Virchow's Archiv, Bd. 61, S. 494.—Schläger: Experiment. Untersuch. über die physiol. Wirkung des Euc. glob. Dissert. Göttingen, 1874.—Bohn: Das Wechselfieber und seine verschiedenen Formen im Kindesalter. Jahrb. f. Kinderheilkunde, N. F., VI., S. 115.—Bérenger-Feraud: De la Fièvre Bilieuse Melanurique des Pays Chauds, 1874.—M'Daniel, E. D.: Hemorrhagic Malarial Fever in Alabama. Transactions Med. Ass. State of Alabama, 1874.—Inman, T.: Malarial Fevers. Brit. Med. Jour., Jan. 2d and Feb. 6th, 1875, pp. 7, 172.—Mosler: Berl. klin. Wochenschr., 1875, S. 668. Deutsches Archiv f. klin. Med., Bd. XV., S. 117.—Finkelstein: Berl. klin. Wochenschr., 1875, S. 494.—London: Anzeiger der K. K. Gesellsch. der Aerzte in Wien, 1875, Sitz. 9, vom 29. Januar.—Hjelt: Virchow's Archiv, Bd. 65, S. 513.—Jerusalimsky: Ueber die physiol. Wirkung des Chinins, 1875.—Kelsch: Archiv. de Physiol., No. 5, 1875.—Binz: Das Chinin, 1876.—Brunner: Die Infections-Krankheiten vom ätiolog. u. prophylact. Standpunkt, 1876.—Sullivan: Med. Times and Gazette, March 11th, 1876.—Von Naegeli: Die niederen Pilze in Infections-Krankheiten, München, 1877.—Love, Wm. J.: Hemorrhagic Malarial Fever, North Car. Med. Jour., Feb., 1878.—Jones, Joseph: Malarial Hæmaturia. New Orleans Med. and Surg. Jour., Feb., 1878.—Aitken, L.: Malarial Fever at Rome. British Med. Jour., April 27th, 1879.—Greene, Wm. A.: Hæmorrhagic Malarial Fever. St. Louis Med. and Surg. Jour., June, 1878.—Norbury, H. F.: Malaria and Ague. Lancet, Oct. 5th, 1878, p. 476.—Smart, Chas.: Mountain Fever and Malarious Waters. Am. Jour. Med. Sciences, January, 1878.—Anderson, Wm.: Iodine in Malarial Fever. Med. Record, Nov. 23d, 1878.—Kelsch, A. et Kiener, P. L.: Des Affections Paludéennes du Foie. Archiv. de Physiol., Sept., 1878.—Reber, Chas. T.: Malaria.—Klebs, E.: Studies on Intermittent Fever and Malaria. Archiv Pathol. und Pharmacol. Leipzig, 1879, XI., pp. 122, 311.—Bérenger-Feraud: Quinine Intoxication and Swamp Poison. Archiv. de Méd. Navale, Paris, 1879, XXXI., 373.—Murchison, C.: Causes of Paroxysmal Fevers. Lancet, 1879, I., p. 617.—Kiener, P. L. et Kelsch, A.: Affections Paludéennes du Foie. Archiv. de Physiol., Nos. 3 et 4, 1879.—Colin, Leon: Maladies Epidémiques. Paris, 1879.—Johnson, W. H.: Malarial Hæmorrhagic Fever. Virginia Med. Monthly, Feb., 1880.—Hoff, J. van Rensselaer: Typho-Malarial Fever, the so-called "Moun-

tain" Fever. Am. Jour. Med. Sciences, January, 1880, p. 38.—BARKER, FORDYCE: Puerperal Malarial Fever. N. Y. Med. Record, Feb. 7th, 1880, p. 158.

The most important contributions to the study of this group of diseases, during the past six years, have related to the nature of their infective cause, and to the melanuric or hæmaturic form of the fever produced by the malarial agent. Prof. von *Nägeli*, the distinguished botanist, has adopted, and presented in scientific form, the hypothesis, previously advanced by *Mitchell*, of Philadelphia, *Salisbury*, of Ohio, *Bartlett*, of Chicago, and others, to the effect that the real infective agent in malaria is a microscopical fungus which by invasion of the blood through the respiratory passages or alimentary canal produces all the phenomena of the various infective diseases. That the active agent is an organized fungus, rather than a gaseous molecule, is thought to be proved by the fact that, if malaria were indefinitely diffusible, as the gases are known to be, it would soon become diluted to such a degree that it would be quite inoperative. The local persistence and comparatively limited diffusion of malaria is believed to favor the hypothesis of a relatively ponderous mass, in contrast with the almost inconceivable minuteness of the gaseous molecule, and yet these particles to which such potency is ascribed are estimated to weigh not more than the thirty-thousand-millionth of a milligramme. Like all other fungi they require air, water, and an appropriate temperature for their rapid multiplication. A freezing temperature renders them inert, but does not destroy them. At the boiling point of water they are disorganized and destroyed; though it is claimed that their spores may support a temperature several degrees above the boiling point without destruction of vitality. These wonderfully minute organic structures are classed in the vegetable kingdom, and are grouped under the general term bacteria. Their universally cellular character is doubtful. *Bergonzini* classifies them as follows:

			Genus.
Bacterium of globular form,			Micrococcus.
Bacteria, linear and straight	short,		Bacterium.
	long,		Bacillus.
Bacteria, linear and spiral	spiral scarcely appparent,		Vibrio.
	spiral very evident	axis short and straight,	Spirillum.
		axis long and folded back,	Spirochaete.

Each one of these genera is subdivided into species of which the whole number does not exceed thirty. Prof. *Nägeli*, however, is not inclined to refer each infective disease to the action of a specific fungus. It is well known that the growth and life-history of these low forms of organized existence vary with the varying conditions of their environment. That these varying conditions determine peculiar modes of virulence in the same fungus is the opinion of the professor. This hypothesis has many facts to recommend it. In the higher orders of the vegetable kingdom, and even in the animal world, we know that plants of the same species may be endowed with poisonous properties which vary with the seasons of the year and the locality of growth. Aconite loses much of its energy when transplanted from its native mountains to the garden of the florist. The centipede and the scorpion of Mexico become harmless when transferred to the south sea islands of the Pacific. In like manner it is possible, though not yet proven, that the ordinary fungi or algæ which vegetate in all places where warmth and moisture favor their growth may, under certain conditions, evolve the violently irritative poison which we know by its effects in cases of poisoning with mouldy bread, stale custards, and similar confectionery. It can hardly be possible, however, that the varied forms of infective disease can be referred to the varying activities of one or only a few of these organisms. It seems more probable that the fungous forms are numerous, and that their effects are limited by the limitations of the human body. A hundred causes may produce inflammation of the skin, but in every instance the inflammatory process is essentially the same. Varieties of climate and of locality must produce a certain correspondence of variety on the part of even the most widely diffused species of fungi. The lack of corresponding variations in the symptoms produced by fungus poisoning in different parts of the world must, therefore, be referred to the determining power of the human tissue cell which is everywhere essentially the same.

A very serious objection to the "fungus theory" of malarial disease lies in the fact that it has been presented in the form of the "parasitic theory" of disease-origin. If malaria consists essentially of fungi or their spores, and if the fever which it originates is caused by their invasion of the blood and their propagation in the tissues, it is impossible to explain the fact that malarial diseases are non-communicable. The evidence of such invasion is also of the most unreliable description. An occasional microscopist announces the discovery of parasitic organisms in the blood of malarial patients; but his observations do not secure the indorsement of those who are best qualified to estimate such researches at their true value. We shall always remain justly skeptical regarding the actual existence of organisms which make no appeal to the senses. But if we turn from organisms to the products of organized bodies, we shall find the difficulties of the problem greatly diminished. Atropia is not an organized substance, it is the product of certain cellular organisms which constitute a portion of the deadly night-shade plant. The introduction of these plant-cells into the blood of an animal produces a fever with one paroxysm. This fever thus produced by infection of the blood is not communicable to a second animal—unless the blood be charged with an amount too great to be appropriated by the cells of the first, in which case the fluids of the first victim may become poisonous to a second, though not communicable by any natural process analogous to ordinary infection or contagion. Now the agent by which the toxic effects are produced is not the cell which proceeds from the plant. It is not the introduction of living cells into the blood which does harm. It is the product, the non-living, unorganized product, of those cells which is the active agent in the production of atropine fever. This illustration may serve to render intelligible the hypothesis of *Bouchardat* (*Rapport sur les progrès de l'hygiène*, Paris, 1867, p. 34; *Annuaire de thérapeutique*, 1866, pp. 299–357), who believes that the best explanation of the behavior of malaria will be secured by regarding it as the product of the vital activity of those low forms of living organisms which abound in desiccating soils. According to this view it should be considered a substance as specifically the product of the fungi which proliferate in certain soils, as atropia is the specific product of the cells of the belladonna plant. This product, set free by the disruption of the parent fungus, passes into the blood chiefly through the medium of the lungs. Its effects in the form of fever and cachexia are the result of a modification of the nutrition of such of the cells of the body as are not actually destroyed by its poisonous energy. This modified nutrition leads to modified function, and this modified function causes a change in many of those bodily reactions which are the result of the varying conditions of heat, light, electricity, and moisture with which the body is in relation. We may thus explain the well-known fact of relapses in the case of malarial subjects many months after their removal from a malarial atmosphere. The restoration of normal structure in such cases has not been complete, though the original malarial poison has long since been eliminated and has ceased to act. A sudden chill, a glass of ice-water, an access of fatigue, the effort of removal itself—a thousand different disturbing causes may alike operate with energy sufficient to disturb the nervous equilibrium of the patient, and to bring about a renewal of the febrile paroxysm. That the intermittent character of the fever need not be referred to the direct and immediate action of the malarial poison itself, but should rather be ascribed to the modified function of the cellular constituents of the body, is rendered probable by the character of the processes which produce the intermittent hectic of septicæmia, in which disease it is not the original infective molecule which directly produces all the subsequent disturbance, but it is the general and secondary alteration of structure and function which sustains the febrile process. The principal difference between a malarial intermittent and a septic intermittent would often seem to consist in the fact that the infective agent in a malarial fever is a substance as heterogeneous to animal tissue as atropia or morphia may be supposed to be, and consequently no more likely to be reproduced by an animal tissue; while the septic molecule, or the typhoid molecule, or any similar agent, being a substance of animal origin and nature, is reproducible by cells like those by which it was originally produced.

Thus far progress has been confined chiefly to the realm of hypothesis. But recently Professor *Klebs* and Professor *Tommasi-Crudeli* (*Archiv für experimentelle Pathologie und Pharm.*, Bd. XI., 1880) have undertaken to supply an experimental basis for the fungoid theory of malaria. They found that after injecting rabbits with an infusion of the soil taken from malarial localities in the vicinity of Rome, the animals exhibited symptoms of intermittent fever, with enlargement of the spleen, and the production of the dark-brown pigment which is so characteristic of the action of malaria upon the red corpuscles of the blood. Injection performed with an infusion of non-malarious soil, from the garden connected with the pathological laboratory at Prague, caused illness, but of a different character. Rabbits which died with symptoms of septic poisoning exhibited a total absence of pigment, with presence of pus, and the large, soft spleen of septicæmia, which was entirely different from the large indurated spleen found in rabbits killed after injection with the malarious infusion. The infusion was then subjected to examination, with the result of discovering a specific fungus, in which the malarial poison was supposed to exist, because the liquid from which these organisms were removed by filtration was no longer nocuous. By cultivation these fungi were isolated and identified as a species of *bacillus*—a species which could also be obtained from the air and the water of the malarious locality. A watery infusion containing bacilli from either of these sources was equally poisonous with infusions of the soil. After injection with these infusions the spleen and the lymphatic glands contained "very small, bright corpuscles which developed, after twenty-four hours in a suitable medium, into threads filled with spores." Similar spores and bacilli were afterwards observed by a Roman physician, Dr. *Marchiafava*, "in the spleen, marrow, and blood of three persons who had died of pernicious fever."

Such experiments are exceedingly interesting, but not wholly convincing. They point out the true method of research, but have not exhausted the subject. That different soils contain different substances of a character noxious to rabbits may be accepted as an established fact. But the circumstance that rabbits are exceedingly sensitive to any form of experiment should not be overlooked. That the poison is confined to the fungi alone, and that it does not also exist as an amorphous ferment in the fluid by which they are supported, cannot be said to be disproved by the results of filtration, because the process of filtration is exceedingly well adapted to disturb the chemical or molecular constitution of an unstable substance. The presence of bacilli, in the bodies of animals thus poisoned by injection, cannot be considered a demonstration of the identity of such bacilli with the poison of malaria, for they are so often discovered in the bodies of animals which have died with other diseases, or which have been merely suffocated. Finally, the *experimentum crucis*, the production of malarial fever in the human subject by injection with infusions like those which were used by Professor *Klebs*, is not yet forthcoming.

Professors *Kelsch* and *Kiener*, of the Military Medical School at Val-de-Grâce, have made a careful study of the changes in the blood and in the liver of Algerian patients suffering with malarial fever, and dying in a state of malarial cachexia. As a result of these researches it is determined that the size of the red corpuscles of the blood becomes enlarged, and their number greatly diminished under the influence of malaria. In the case of a patient with a quotidian intermittent, there was in twenty-four hours a loss of more than one million red corpuscles per cubic millimetre of blood. This great destruction is evidently the cause of the increase of pigment, which loads the white corpuscles of the blood as well as the cells which compose the liver, spleen, and other organs of the body. In the profound anæmia caused by malarial poisoning, the number of red corpuscles per cubic millimetre may fall from five millions to even less than a million and a half.

In the liver, under the influence of malarial hyperæmia, the changes may be identical with those which are produced by hyperæmia from any cause. There is dilatation of the capillaries, with endothelial proliferation in their walls. Leucocytes and wandering cells—often charged with pigment—accumulate in these vessels. The hepatic cells become hypertrophied and somewhat increased in number. There is distention of the lymphatic spaces, and sometimes a commencement of annular cirrhosis. These alterations in a malarial liver are carried to their highest degree, and tend to become chronic.

In miliary inflammation of the parenchyma of the liver there is a clinical history which cannot be distinguished from that of simple malarial hypertrophy of the organ. The general changes in the capillaries and in the cellular structure of the liver are the same as in simple hyperæmia. But the cellular hyperplasia predominates over the other changes to a degree which constitutes a distinct morbid process. The surface of the enlarged organ is roughened by little elevations, varying from the size of the head of a pin to a hemp-seed. These are the so-called miliary granulations, which are also found throughout the organ. These granulations are the result of an irregular increase in the glandular elements of the lobules, so that, while in one portion they are greatly multiplied, the contiguous portions are condensed and atrophied by pressure. If the glandular elements continue to increase, the vascular network is encroached upon to a degree which threatens necrobiosis of the part. As a consequence of the hindrance to the circulation thus established, the blood which is forced into the liver under the influence of malarial congestions tends to accumulate in other portions of the vascular network, causing compression and corresponding atrophy of the adjacent glandular elements. These disappear entirely at certain points, while at others they seem to be transformed into connective tissue.

Chronic or nodular parenchymatous hepatitis is characterized by diminution of the volume of the liver, and death is often preceded by dropsy, icterus, petechiæ, and somnolence—symptoms which differ from those produced by ordinary degeneration of the organ, chiefly in the febrile movement by which they are preceded and accompanied. The histological appearances ally the disease to miliary hepatitis, but the nodular formations are more extensive, and becoming crowded together, tend to a variety of results which may be ranked in two principal groups, characterized on the one hand by fatty, colloid, caseous, or puriform softening of the central portions of the nodules, and, on the other hand, by the transformation of the inflamed glandular tissue into embryonic connective tissue. In this last case it would seem as if the epithelial gland-cells, by freeing themselves of their protoplasm, become reduced to the condition of free nuclei, which then assume the appearance and behavior of embryonic connective-tissue cells. It is through the formation of this new tissue between the nodular masses that this form of hepatitis is finally differentiated from miliary hepatitis, in which the miliary granulations are separated from each other by bands of hyperæmia or condensed gland-substance alone.

Following close upon the steps of parenchymatous hepatitis are two forms of cirrhosis—annular and diffuse. The annular form surrounds the nodules which are composed of several lobules or parts of lobules, and differs from the ordinary form of cirrhosis in the fact that, being developed from the glandular elements of the parenchyma, it is not confined to the territory of the capsule of *Glisson*. Pure, extra-lobular cirrhosis is rarely found in the endemic forms of hepatitis, caused by the malaria of Algeria. Ascites is characteristic of malarial cirrhosis, as well as of the classic form of the disease, but its course is more rapid, and it is associated with a nearly continuous febrile movement.

Malarial hæmaturia, or melanuric fever, is a variety of malarial fever already described in this cyclopædia, but it has recently been carefully studied in Senegal, by *Bérenger-Feraud*, and in the southern United States of America, by *Joseph Jones* and others. It is a form of malarial fever characterized by complete jaundice, persistent vomiting of green biliary matter, and a peculiar brownish color of the urine. In Senegal, this color is ascribed to the presence of bile and biliary pigment in the urine, to the almost constant exclusion of blood or hæmatin. But the American physicians have usually encountered an abundant hemorrhage from the kidneys in their cases of the disease. *Jones* has detected the presence of albumen, colored blood-corpuscles, excretory cells of the kidneys, and casts of the tubuli uriniferi. These casts are often impacted with red blood-corpuscles. The disease usually occurs in cases previously exhausted by malarial cachexia. It is ushered in by exhausting chills of an intermittent type. Headache, nausea, vomiting, and melanuria complete the clinical picture. The course of the fever may be intermittent, remittent, or continued. A sensation of weight and tenderness in the infra-diaphragmatic region is usually experienced. In severe cases there may be a bilious diarrhœa. Favorable cases terminate with gentle perspiration and gradual subsidence of the symptoms. Fatal cases often terminate in collapse with suppression of urine, convulsions, delirium, and coma. The duration of the disease is exceedingly variable, proving fatal in two or three days, or lingering for many weeks. Mild cases usually recover. In severe forms of the disease the mortality is about twenty per cent. In the worst cases death is the rule. After death the pathological changes are those of intense hyperæmia of the liver, spleen, and kidneys. The gall-bladder is distended with bile, a fluid which is universally present in the vomit of malarial hæmaturia as it is universally absent in the black vomit of yellow fever. The only diseases with which malarial hæmaturia can be confounded are acute atrophy of the liver and yellow fever. In acute atrophy there is absence of fever, diminution of the volume of the liver, negative action of quinia, and no relations with malaria. In yellow fever the clinical features of the paroxysm, the absence of bile in the final stage of black vomit, the diminution of bile in the gall-bladder and biliary passages, the presence of yellow granular and oleaginous matters without blood-corpuscles in the tubuli uriniferi, are sufficient to distinguish the disease from hæmaturia. Analysis of the blood by Dr. *Jones* exhibited an increase of pigment and the presence of urea and the biliary acids in the blood. The red blood-corpuscles were diminished in number, and the fibrin was increased.

1000 *Parts of Blood contained:*

Water,	820.50
Solid matter, including fixed salts,	179.50
Fixed saline constituents,	7.18

1000 *Parts of Serum contained:*

Water,	902.66
Solid matter, including fixed salts,	97.34
Fixed saline constituents,	5.77

1000 *Parts of Blood contained:*

Water,	820.50
Dried blood-corpuscles,	86.47
Fibrin,	4.50
Albumen and extractive matter,	88.53
Fixed saline matter,	7.18

This increase of fibrin is considered evidence of an inflammatory condition of the body, and an indication for a certain degree of antiphlogistic energy in the treatment of the disease.

Analysis of the urine indicates an increase of urea, urates, and phosphates during the progress and increment of the disease.

1500 *cc. of Urine passed during* 24 *hours contained:*

Urea,	528.52	grains.
Uric acid,	18.72	"
Phosphoric acid,	120.12	"
Sulphuric acid,	51.38	"
Chloride of sodium,	24.02	"
Blood casts, albumen, and bile,	present.	

Reaction, acid.
Specific gravity, 1014.

In the treatment of the disease, *Jones* inclines to the employment of local depletion, with mercurials in small and continuous doses, to be associated with sulphate of quinia in considerable quantity, and rhubarb, aloes, and colocynth if the bowels are sluggish. A tonic treatment with nitro-muriatic acid or tincture of the perchloride of iron with sulphate of quinia, must be continued during the period of convalescence. Change of climate, if possible, is imperative. In the African variety of the disease, the irritative phenomena of its inception are treated with evacuants and with opiates. Small doses of opium are given every hour until the stomach will tolerate the administration of quinia which is then exhibited in large and frequent doses. The use of mercurials is discountenanced.

Fordyce Barker, of New York, has lately redirected the attention of the profession to the increased liability of women to the effects of malaria immediately after confinement. Puerperal malarial fever may be declared at any period after delivery until convalescence is complete. The outset of the disease is abrupt, marked by chills, and by a temperature of one or two degrees higher than the temperature of any other form of puerperal fever. The degree of prostration is greater than is usual in other diseases to which the parturient female is liable. The intermissions or remissions of the fever follow the usual course of a malarial fever, and a fall of temperature is accompanied by a corresponding decline of other symptoms—a fact which assists in the differentiation of this disease from puerperal septicæmia. Recovery is the rule. The treatment coincides with that which is found most useful in other forms of malarial fever.

The so-called "mountain fever," of the Rocky Mountain Territories of the United States of America, has received careful attention on the part of the medical officers of the United States Army. It has by many observers been considered as a form of malarial fever, ascribed to the use of "aqua-malarial" waters; but it is now fully identified as typhoid fever.

DENGUE.

BIBLIOGRAPHY.

VERCHERE, A. M.: Report on Dengue at Calcutta, 1872. Indian Med. Gaz., Calcutta, 1879, XIV., 91-121.—MORICE: De la Dengue et de sa Distrib. géogr., Paris, 1875.—LACAZE, M.: Dengue. Bul. Soc. Méd. Practicale, Paris, 1878-79, pp. 98-106.—COLIN, LEON: Traité des Maladies Epidémiques, Paris, 1879.

The infective character of this disease is now established. Arising in the Antilles, it follows the track of commerce throughout the West Indies and along the coast of the Caribbean Sea. The same facts of propagation have been observed upon the islands and the shores of the Indian Ocean. It occurs in the Red Sea, and has been traced among the islands of the Pacific. It ranks next to influenza in the rapidity and in the extent of its diffusion. Its epidemics spread regularly from their points of origin, and subside gradually. Sporadic cases often follow an epidemic, even a year later. The disease occasionally appears in sporadic form in the maritime cities of the north. The incubation of the disease is very brief—four or five days, or even less. Generally, it is only a few hours. Each case becomes infective soon after its commencement, so that the disease is propagated with great rapidity, but in Samarang, in 1872–73, its diffusion was at so moderate a pace that many doubted its communicability. Long intercourse with the sick confers no immunity. Doctors and nurses share the illness of their patients. The domestic animals may also be affected. Cattle in India, during the prevalence of an epidemic, manifested symptoms of joint-disease, continuing for three or four days. Cows and horses were also affected in the same way. In Rangoon, during the epidemic of 1872, cats and dogs were attacked with rheumatism.

There is direct evidence that the disease may be conveyed in clothing and bedding. It has thus been carried from Aden to India and Java, and from Central America to Spain. It is often diffused by ships, troops, and emigrants, just as cholera, typhus fever, and small-pox are spread by similar agencies. Heat and moisture seem to favor its epidemic prevalence, but it may be propagated to a certain extent without regard to such agencies.

The first stage of the disease is characterized by fever and a painful swelling of the joints like that in rheumatism. There is also a slight and variable exanthem, and a temperature of 106 to 107.6° F. After two or three days, sometimes even later, there is subsidence of the temperature and abatement of the febrile movement, though the temperature often remains slightly above the normal degree. After a remission, which generally lasts one or two days, the fever is renewed with a second eruption which varies in its character. During the remission there is profuse sweating with an odor like that of decayed straw. Diarrhœa or epistaxis may also occur. The terminal exanthem is followed by desquamation. In severe forms of the disease, there is great suffering with severe supra-orbital headache, pains in the ligaments of the joints and in the muscles. Conjunctivitis, coryza, angina faucium, and bronchitis are often experienced. Wakefulness, debility, nervous prostration, partial anæsthesia or hyperæsthesia of the skin, gastric embarrassment, and constipation are common symptoms during the first stage. The inguinal lymphatics become swelled and painful in certain cases. Cardiac affections are not uncommon. The prognosis is almost always favorable, though relapses are frequent. They may occur a week or longer after the subsidence of the original attack, which lasts only five or six days. The joint affections may linger for weeks or even months. Convalescence is usually slow, and the establishment of vigor requires a long period of time.

The following notes of a case, reported by *Slaughter*, represent the typical course of this disease :

First day.—Temperature 104–105° F. Pulse frequent. Pain in back and joints, headache, vomiting, great prostration. Conjunctivæ injected, skin dry. Delirium, sleeplessness, partial anæsthesia. Urine scanty. Tongue moist, its papillæ red.

Second day.—Temperature 101–103° F. Pulse frequent. Pain in the back, rheumatic pains in the limbs, headache. Profuse perspiration of a peculiar odor. An exanthem upon the nates and extremities. Face red, eyes injected. Urine increased, urates precipitated. Tongue irritable, breath offensive. Memory diminished.

Third day.—Temperature 97.7–99.5° F. Pulse weak, small, and frequent. Pains in joints and face. Headache and back-ache last longer. Profuse perspiration. Exanthem prominent. Lymphatics swelled and painful. Less weakness. Redness of face and conjunctivæ diminished. Memory still defective. Urates precipitated, and urinary sediments with a pungent odor. Tongue and mucous membrane sensitive. Breath offensive.

Fourth day.—Temperature 97–99° F. Pulse normal. General improvement. Face and eyes better. Pains in limbs still troublesome, head and back less painful. Skin moist. Exanthem less brilliant. Appetite returning. Urine strong, urates abundant. Lymphatic vessels still swelled and painful. Memory deficient. Tongue tremulous and sensitive at the edges. Fauces slightly sensitive.

Fifth day.—Temperature and pulse normal. Feeble, but growing convalescent. Pain still experienced in the limbs. Exanthem disappeared. Appetite good. Memory returning. Lymphatics less painful. Strong smelling urine with urates.

Sixth day.—Temperature normal. The severer symptoms and local pains disappear.

Seventh day.—Weak. Anæmic.

Eighth day.—Full recovery.

Symptoms of the Disease.—Dengue commences suddenly, a few hours only after exposure. It is often ushered in by a severe chill. In children this may be replaced by convulsions, or by hysteria in women and nervous persons. Sometimes the attack is preceded by several days of discomfort. The joints are commonly affected. There are general pains and cramps so that the feet cannot be placed on the ground. The phalangeal articulations are often affected in such a manner that the patient is led to suspect the presence of some other disease. From the fingers the swelling may advance in an orderly manner to other joints. Often, after the first violent paroxysm, the joints may remain affected for a considerable time. Sometimes, only a limited number—four or five—of the joints are involved. The swelling may be confined to the fingers alone. It is prone to attack those joints which have been enfeebled by previous injury. The swelling is considerable, rendering movement very painful, hence the peculiar attitude which has given the disease its name, dandy fever. Serous infiltration of certain joints, and reddening of the crucial ligaments of the knee have been observed.

The muscles and fasciæ become involved, the sinews are painful; boring and lancinating pains are felt in the knees, and all the limbs are stiffened. Often, especially in children, the abdominal walls are painful. These acute symptoms continue one or two days, but the stiffness and crippling of the limbs may continue for a longer time.

Nervous Phenomena.—The pain in the head and back resembles the pain experienced in variola. It continues for two or three days in the frontal and orbital regions. The intellectual faculties are depressed throughout the whole course of the disease. The patient is restless, sleepless, or troubled with frightful dreams, if he falls asleep. These nervous symptoms often continue after the conclusion of the fever. There is extreme prostration, and the condition of anæmia and debility frequently continues long after the establishment of convalescence. General emaciation also is often observed. Convulsions of an epileptiform character sometimes accompany the disease. A condition of coma may continue for a day at a time. Children often become wildly delirious. Respiratory spasm, continuing for two or three minutes, has been noted in at least one case, with recovery at last.

Cutaneous Phenomena.—The initial exanthem is observed in one-half or two-thirds of the cases. It generally begins as an efflorescence covering the entire body, or in scarlet blotches upon the face and extending to the trunk. It appears less frequently upon the extremities. Sometimes the rash appears only upon an arm or a leg. The conjunctivæ are vividly injected; the eyelids and the face swell. The skin is usually hot and dry; a red line follows the track of a finger-nail drawn over the surface. Perversions of sensation also occur. The initial exanthem usually continues five or six hours—sometimes for twenty-four hours. Its disappearance, with a very trifling desquamation, commonly coincides with the subsidence of the joint affection. Relapses sometimes occur. The terminal exanthem makes its appearance on the third or fourth day of the disease. It is more uniformly present than the initial eruption, occurring in at least two-thirds of the cases. It is polymorphous in its character, resembling measles, scarlet-fever, or roseola. Vesicles and pemphigoid blebs are sometimes present. The eruption of dengue, though closely resembling the exanthem of measles or of scarlet-fever, does not commence upon the face, but about the knee, the elbow, or the hand, while the breast may often remain free from large spots. Not unfrequently the terminal exanthem persists only for an hour; and on the contrary it may continue two or three days. Dengue sometimes counterfeits an intermittent fever, and is benefited by quinine. Relapses of the eruption often assume the form of urticaria. Such a relapse may take place at the expiration of the second day. The hyperæmia of the skin may even result in ecchymosis. European and fair-skinned persons exhibit a blush upon the palm of the hands and the soles of the feet. Desquamation of the cuticle often takes place eight or ten days after the establishment of convalescence. It resembles the similar process after measles and scarlet-fever, but is sometimes more penetrating in its effects upon the skin. Perspiration accompanies the first subsidence of the temperature, and is characterized by a peculiarly specific odor. Though the lymphatics of the neck and groin are swelled, they do not suppurate.

Febrile Phenomena.—During the initial paroxysm there is a temperature of 104° F., continuing one or two days, with evening exacerbations and morning remissions. The subsidence of temperature is sudden, and accompanied with copious perspiration. This is sometimes prolonged for twenty-four or thirty-six hours. During the next four or five days the temperature may be less than normal. In New Orleans, during the epidemic of 1874, Dr. *D'Aquin* found the temperature a little above the normal degree—about 100° F.—throughout this stage of the disease. The pulse ranges from 90 to 120; in the first stage it is full, hard, and tense; during the second stage it is small and weak. Its rate does not correspond with variations of the temperature. There may be a pericardial murmur, which disappears at the close of the initial paroxysm. The urine is slightly lessened in quantity, and heightened in color; its sediment varies from white to flesh-color. Phosphates appear at convalescence. The specific gravity of the urine is below the normal figure; its reaction may be slightly acid or neutral. Examination of the blood reveals the presence of numerous small round corpuscles, which stain carmine-red when treated with hypo-osmic acid; they make their appearance between the third and the sixth day of the disease. Although present in other exanthematous diseases, they occur with such regularity in dengue that they may be considered almost differential and diagnostic. They have been regarded by certain observers as analogous with organic germs. The mucous membrane shares in the general disorder; there is conjunctivitis, coryza, and epistaxis. The mucous membrane of the mouth, fauces, and tonsils may swell and become ulcerated. The submaxillary glands enlarge, and there is a profuse discharge of saliva, with a fœtid condition of the breath. Laryngeal and bronchial catarrh may be established, and pulmonary complications may follow. The gastro-intestinal mucous membrane is less likely to be thus invaded than the respiratory passages. The movements of the bowels become irregular or may be arrested. Diarrhœa, with copious, watery stools charged with intestinal epithelium, sometimes is present. There is loss of appetite, and considerable thirst. Nausea and vomiting are frequent, and among children the vomited matter may contain blood. Such cases are likely to prove fatal. In countries where yellow fever prevails, dengue may commence with jaundice and black vomit. The tongue is broad, its edges are red, and the papillæ are prominent. In females the disease may excite menorrhagia and produce abortion.

Relapses occur about ten or eleven days after the commencement of the disease. By certain writers these have been ascribed to the influence of malaria.

Stages of the Disease.—*First.* In more than half the cases the invasion of the disease is sudden. This stage may continue with varied symptoms for three or four days. *Second.* The pyretic stage lasts twenty-four or forty-eight hours. *Third.* The exanthematous or apyretic stage, extending from the initial paroxysm to the terminal exanthem, lasts from a few hours to two days. *Fourth.* Stage of relapse, which is of short duration. *Fifth.* Stage of convalescence, lasting three or four days. The entire average duration of the disease is from five and a half to seven days.

Prognosis.—In the Anglo-Indian army (1872) only 1 in 7,435 died. Out of 8,069 cases elsewhere reported, 25 died, of whom seven were men, one was a woman, and seventeen were children. The highest average mortality is only one-half of one per-cent. Death generally results from collapse with choleraic symptoms, or during the period of high temperature.

Diagnosis.—The diseases with which dengue may be confounded are: 1st. *Thermal fever,* in which, however, the cerebral symptoms are more pronounced than in dengue, and the joint symptoms are absent. 2d. *Rheumatic polyarthritis.* The joint affections are quite similar, but in dengue the swelling occurs more suddenly, and there is a more rapid subsidence of temperature after the cessation of severe pain. 3d. *Scarlatina,* a disease which may be recognized by the fact that pain in the joints is most severe after the subsidence of the eruption. The pulse of scarlet-fever is more frequent, and the exanthem disappears more gradually than the eruption of dengue. Albuminuria is not one of the sequelæ of dengue. 4th. *Measles* may be distinguished from this disease by the absence of joint affections, and by the greater severity of the catarrhal symptoms. 5th. *Influenza* resembles dengue in its wide diffusion and its epidemic character. Its mode of invasion and its most characteristic symptoms resemble corresponding symptoms in dengue; but it is not communicable, and does not involve the joints. 6th. *Erythema exsudativum multiforme.* Of this disease epidemics have occurred in Paris (1828), and in Frankfort, in the Turkish armies (1852), and in Bosnia (1858). Its symptoms closely resemble those of dengue, but it differs in the degree of contagion—when both are epidemic, dengue is the more contagious. The course of symptoms falls into a succession of typical periods in dengue, while in erythema the phenomena assume a variable order. Dengue is a specific disease, produced by a definite poison invading the system and developed within the organism—a proposition which cannot be affirmed of erythema, a disease in which the initial lesion is due to some chemical or mechanical action upon the mucous membranes. The transitory forms of erythema are maintained by some form of mechanical or thermal energy acting upon the skin. In epidemics of the disease the presence of a specific poison may be suspected.

Therapeutic Measures.—At the commencement of the disease the alimentary canal should be evacuated by the use of an emetic and an enema. If the temperature reaches 107° F., cold baths should be employed. During the exanthematous stage, diaphoretics may be exhibited, and the body should be protected from cold. For severe headache, hot foot-baths, mustard poultices, leeches, and atropia by hypodermic injection will be of service. Painful joints should be rubbed with camphorated oil, laudanum, or chloroform liniment. Sulphur baths and electricity are recommended; also iodide of potassium in small doses. Children with convulsions should be made to inhale chloroform, and may be treated with cold shower-baths, bromide of potassium, extract of cicuta, and asafœtida enemas. The English rely upon supporting treatment with iron and quinine. Some are disposed to consider quinine as a prophylactic. A quarantine of four days is often enforced, and is theoretically useful.

ERYSIPELAS.

BIBLIOGRAPHY.

YEAR 1874.

HUTCHINSON: On the Hospital Plagues. Brit. Med. Jour., p. 161.—ERICHSEN: On Hospitalism. Ib. p. 133.—TIBBITS: On Erysip. in Hosp. and General Practice. Lancet, I., p. 832.—GLEASON: Boston Med. and Surg. Jour., No. 20.—MAPOTHER: On the Nature of Erys. Med. Press and Circular, 21st of Jan.—GREENE: Ib., May 27th.—STEWART: Erys. in Puerperal Condition. Brit. Med. Jour., p. 305.—REID: Erys. During Puerperal Condition. Ib., p. 646.—MINOR: Erys. and Childbed Fever (Monograph). Cincinnati.—WOJLANSKI: Considérations sur Erys. (Thesis).—BOHN: Impf-E. und seine Bedeutung zur E.-Lehre. Jahrb. f. Kinderhlkde, VIII., S. 1.—LÜHE: Dtschs. Arch. f. klin. Med., XV., S. 99.—PERROUD: Jour. de Bruxell., LVIII., p. 532.—HILLER: Berl. klin. Wochenschr., XI., S. 48.—LUKOMSKY: Unters. üb. E. Virch. Archiv, 60, S. 418.—RENAUT: Arch. de Phys., 2, I., p. 234.—LORDEREAU: Arch. gén., XXIII., p. 276.—BROUARDEL: Gaz. d. Hôp., 5, 6.—FLAMMARION: ib., 12.—CADIAT: Jour. de l'Anatomie et Physiol., X., p. 410.—FREDET: Case of E. with Gangrene. Gaz. d. Hôp., 87, 88.—VINCENT: Erys. in the Newly-Born, Ib., 96.—LUTZ: Ib., Deutsch. Arch. f. kl. Med., XIV., S. 137.—MAYER: Dtsch. Ztschr. f. prakt. Med., 12, 13.—DUPLAY: Phlegmonous Erys. Arch. gén. de Méd., Dec.—ALLAN: Idiopath. Facial E. Med. Times and Gaz., I., p. 586.—HESSE: The same. Schmidt's Jahrb., 162, S. 248 —MAURIAC: Erys. Syph., Paris.—GRIVET: Etude Clin. de l'influence de l'E. sur le Lupus. Thèse, Paris.—ROSSMUND: Chinin bei E. d. Kinder. Deutsche K., 7.—MUNSELL: Use of Tinct. Ferri Sesquichl. Phila. Med. and Surg. Rep., Dec. 26th, 1874.—SENSTIUS: Berl. Diss. (Carbols. in 3 Fällen ohne Erfolg).—HALL: Treatment of E. with Aspiration. Lancet, I., April 14th, 1874.—THORESEN: Nord. Med. Ark., VI., Nr. 22.

YEAR 1875.

HOHN: Handb. d. Vaccinat. Leipzig (S. 167 ff.).—HATHERLEY: Brit. Med. Jour., April 17th.—SQUIRE: Ib., May 22d.—MINNICH: E. Migrans von bes. Intensität. Wien. med. Presse, No. 5.—BIERBAUM: E. Serpens S. Univers. bei Neugeborenen u. Säuglingen. Dtsch. Klin., 8, 12.—JACOBS: Presse méd. belg., 121.—TORDAY: Ueb. Impf. E. Pest. Med. Presse, XI., 23, 24.—NIPPOLD: Carbols. subcutan. Winckel's Studien, S. 186.—MUNSELL: Phila. Med. and Surg. Reporter, XXXII., p. 203.—BOCKEL: (Gaz. méd. de Strassb.) ärtzl. Mitth. aus Baden, XXIX., 12.—BAY: Hosp. Tidende, 2 R., II., 27.—FOSTER: Med. Times and Gaz., April 3d.—LE MONNIER: Americ. Jour., XXXVII., p. 294, Jan.—ZUDZER: (Verbreitung des E.) Ueber städt. Grundlagen für die Hygieine und specielle Aetiologie. Beitr. z. Med.

Statis., I.—Zerker: Epid. von Erys. in einer Irrenanstalt. Allgem. Ztschr. f. Psych., Bd. 32, S. 28.—Davies, K.: Erys. in Puerperal F. Brit. Med. Jour., I., p. 362.—Wells, Spencer: Ib., I., 501 II., 105.—Ib.: Erys. in Radcliffe Infirmary, Oxford, ib., 641.—Smith: Lancet, p. 187.—St. Phillip: Gaz. méd. de Bordeaux, Oct. 5th.—Morbieu: Récidive. Union méd., III.—Hug: Bayr. ärzt. Int. Bl., Nr. 49. (Impfrothlauf).—Schneider: Berliner klin. Wochenschr., 525 (desgl. ohne Erfolg angew.).—Foster: Med. Times and Gazette, 374.—Calvy: Union Méd., III. (Collod.).

YEAR 1876.

Sinnhold: Jahrb. f. Kinderk., S. 383.—Meinert: Deutsche med. Wochenschr., S. 35 (Impferysipel.).—Deahna: Viertelj. f. Dermatol., S. 57.—v. Rothmund: Bayr. ärzt. Int. Bl., Nr. 29.—Bell: Edinburgh Med. Jour., p. 97 (Tinct. ferri mur.).—Fontenoy: Hosp. Tid., III., 15.—Bellien: Mosk. Med. Ztg., Nr. 1, 3. —Takacz: Pest. med. chir. Presse, Nr. 29.—Cavazzini: Gaz. med. Ital.—Alvarenga: France méd. 6. Janv.

YEAR 1877.

Crocker, John M.: Erys. and Puerperal Fever. Boston Med. and Surg. Jour., No. 5, August 2d.

YEAR 1878.

Erichsen: Erysipelas of the Scalp. Lancet, January 26th, p. 115.—Mendörfer, J.: Zur Pathologie und Therapie der Erysipelas. Wien. med. Presse, 1878, XIX., 1318–1321.—Hardy: De l'érysipèle. Union méd., Paris, 1878, XXVI., pp. 377, 401, 473, 462.—Davezac: De l'érysipèle de la Face. J. de méd. de Bourdeaux, 1878–79, I., 252.—Whitmire, J. S.: Erysipelas, Carbolic Acid Injections. Chicago Med. Journ., March, 1878, p. 239.—Caldwell, W. S.: Erysipelas. Ibid., Dec., 1878, p. 610.—Tidmanns, H.: Experimentelle und anatomische Untersuchungen über Erysipelas. Verhandl. d. deutsch. Gesellsch. f. Chir., Berl., 1878, VII., pt. 2, 165, 247, 1 pl.

YEAR 1879.

Ibid.: Experimentelle und anatomische Untersuchungen über Erysipelas. Archiv f. klin. Chir., Berl., 1879, XXIII., 437, 519, 1 pl.—Blin, L.: De la contagion de l'érysipèle. Saint Quentin, 1879, 8vo.—De G. Griffith, George: Unity of the Poisons of Scarlatina, Puerperal F., Typhoid, Diphtheria, and Erysipelas. Med. Press and Circular, London, 1879, XXVII., 315.—Verneuil: Erysipelas. Paris méd., 1879, V., 329–332.—Cheurlin, Ernest: Etude clinique et pathologique des rapports de la péritonite avec l'érysipèle. Paris, 1879, 52 pp., 4to, No. 97.—Morienvalle, D.: Contribution à l'étude de l'érysipèle du Pharynx. Paris, 1879, 50 pp. 4to, No. 97. Colin, Leon: Trait. des Malad. Epidem., Paris, 1879.—Pepper, Wm.: Erysipelas. Med. and Surg. Reporter, Feb. 28th, 1880.

The relation between erysipelas and child-bed fever is now fully established. Doubts regarding the reality of such a connection have hitherto rested upon a failure to distinguish between the infective degrees of virulence which separate traumatic erysipelas from ordinary facial or cutaneous erysipelas. The former variety of the disease is exceedingly virulent, while the latter form is fairly benign. Parturient women who are exposed to the virus of the former contract puerperal fever, but remain healthy when attended by physicians and nurses who have the care of cases of facial erysipelas. Women who are subject to attacks of cutaneous erysipelas, or who belong to families that exhibit a predisposition to the disease, are liable to puerperal fever after child-birth. The inflammation is very likely in such cases to spread rapidly to the peritoneum, with an almost inevitably fatal result. The sudden development of a febrile movement with swelling of the spleen after delivery, are circumstances which usher in the attack of puerperal erysipelas. It is usually confined to the genital organs, and to the peritoneum, but it may appear concurrently upon the face or other external portion of the body.

The treatment of erysipelas presents no special advance. The use of carbolic acid, hypodermically—suggested by the supposed dependence of the disease upon the development of parasitic fungi within the tissues—is now abandoned. The progress of the disease was scarcely modified, and abscesses were not infrequently formed at the points of injection.

CEREBRO-SPINAL MENINGITIS.

BY

J. LEWIS SMITH, M.D.,

OF NEW YORK,

CLINICAL PROFESSOR OF DISEASES OF CHILDREN IN BELLEVUE HOSPITAL MEDICAL COLLEGE.

CEREBRO-SPINAL MENINGITIS.

BIBLIOGRAPHY.

Verardini: Bullet. delle Sc. med. di Bologna, 1874, Nobr., p. 321.—Amoroso: (Tommasini, Neapel) Il Morgagni, 1874, Disp. IV., p. 241.—Heiberg: Hospitals Tidende R. II., 1, p. 737, 1874.—J. Lewis Smith: Cerebro-Spinal Fever, with Facts and Statistics of the recent Epidemic in New York City. Amer. Jour. of Med. Sci., Oct., 1873.—Clymer: Epid. Cerebro-Spinal Meningitis, with a map of the City of New York, showing the Localities of the Disease in the Epidemic of 1872. Phil., 1873.—Upham: Some Additional Reflections upon the late Epidemic of Cerebro-Spinal Meningitis. Bost. Med. and Surg. Jour., Sept. 3d, 1874.—Crooks: Phil. Med. and Surg. Reporter, Jan. 24th, p. 86, 1874.—Thomson, W. H.: On the Treatment of Cerebro-Spinal Meningitis. Phil. Med. and Surg. Reporter, 1875, p. 269, April 3d.—Faulhaber, F.: Ueber Meningitis Cerebro-Spinalis. Dissert. inaug., Berlin, 1875.—V. Reichmann: Ueber das sporadische Auftreten der M. c. sp. Epid. Nach Beobachtungen in Tūbingen 6, Fälle. Dissert inaugur. Tūbingen, 1876.—Moos: Archiv f. Augen- und Ohrenheilkunde, III., 1, S. 76, 1874.—Tr. M. and Chir. Fac., Baltimore, Md., 1879, LXXXI., 116–121.—O'Neill, E. D.: Simulative Cerebro-Spinal Meningitis, with Abnormally High Temperature. Med. Press and Circ., London, 1879, N. S. XXVIII., 131.—Perkahl, Z.: Zapalenie epidemiczne powlok Mózgordzeniowych (Meningitis cerebro-spinalis epidem.). Gaz. lek., Warsawa, 1879, XXVII., 21-24.—Russell, I.: Cerebro-Spinal Meningitis. Birmingh. M. Rev., 1879, VIII., 51-64.—Rankin, I. D.: Lecture on Cerebro-Spinal Meningitis. Texas Med. Jour., April, 1878.—E. B. Knight: A Case. N. E. Med. Gaz., July, 1878.—D'Ans: Typhus Cérébro-Spinal Epidémique: Extrait du Rapport Méd. (2e Semestre, 1878) de l'Hôpital Militaire de Diest. Arch. Méd. Belges, Brux., 1879, 3s., XVI., 185–192.—Dubar, L.: Meningite Cérébro-Spinale Tuberculeuse. Progrès méd., Paris, 1879, VII., 834.—Oltuszewski: Zapalenie opon Mózgo-rdzenio-wych (meningitis cerebro-spinalis). Gaz. lek., Warszawa, 1879, XXVII., 94.—Van Bellinghen: Meningite Cérébro-Rachidienne, suite d'Accès Hystériques; Anti-phlogistiques; Calomel; Torsion abdominale; Guérison. Presse méd. Belge, Brux., XXXI., 353–355.—Warschauer, I.: Ueber Meningitis Cerebro-Spinalis Epidemica. Allg. Wien. Med. Ztg., 1879, XXIV., 481, 498.—Hunt, I. W.: Case of Purulent Cerebral and Spinal Meningitis, No Symptoms; Sudden Death by Coma.—Brain, I.: Neurol. London, 1879–80, II., 409-412.—Hennig, C.: Ueber Meningitis Cerebrospinalis Epidemica, Deutsche med. Wchnschr., Berl., 1879, V., 380–382.—Oeller, I. N.: Retinitis und Cyclitis Suppurativa bei Cerebro-Spinal Meningitis. Arch. f. Augenh., Wiesb., 1879, VIII., 357–378.—Ohr, C. H.: Cerebro-Spinal Meningitis; Spotted Fever.

Cerebro-spinal fever, or, as it is more commonly known, cerebro-spinal meningitis, is regarded by pathologists as a constitutional disease with local manifestations which pertain chiefly to the cerebro-spinal axis. It occurs as an epidemic in various and distant localities over limited areas, and for a limited period. It is rarely, perhaps never, established as an endemic like diphtheria, though isolated cases sometimes occur which are not distinguishable in symptoms and lesions from those occurring during an epidemic. Its cause, subtle and undetermined, seems to sustain an etiological relation, also, to certain inflammatory diseases not affecting the cerebro-spinal axis, during the time when epidemics of the meningitis prevail. Thus in this country, during the occurrence of cerebro-spinal meningitis between 1811 and 1815, pharyngeal and pneumonic inflammations were common; and *Webber* in his prize essay describes an exceptional form of this disease which he designates pneumonic, in which the cerebro-spinal axis is not involved, or is but slightly involved, and the chief local manifestation is in the lungs. During the epidemic in New York City, in the spring of 1872, there were 1,707 deaths from diseases of the respiratory apparatus exclusive of tuberculosis in the four months when meningitis was most prevalent, while there were only 1,336 deaths from the same diseases in the remaining eight months.

CAUSE.

This is obscure, though certain predisposing influences, as anti-hygienic conditions, are well established. The cause does not apparently emanate from the soil, for a majority of the epidemics occur in the winter when the ground is frozen, and in localities, often far apart, having different geological formations and altitudes, while neighboring localities having the same kind of soil escape. Nor do facts justify the belief that the specific principle resides in the food, as some observers have supposed, for of two adjacent localities, in which the food of the inhabitants is the same, one is visited by the epidemic, while the other escapes. Infants nourished entirely by breast-milk are liable to be attacked, and so are certain animals, like the horse, whose food is entirely different from that of man. Moreover, careful examinations of the cereals and other food by scientific observers have hitherto failed to discover any uniform noxious principles, or any substance which would be likely to cause a malady of this nature. There can be little doubt, therefore, that the cause is atmospheric.

Of the predisposing causes, anti-hygienic conditions hold a prominent place. Soldiers in barracks, and the city poor in tenement houses, suffer most severely. During the epidemic which visited New York in 1872, unusually severe or multiple cases occurred for the most part where there were obvious anti-hygienic conditions, as crowding, or a filthy state of the domiciles or bedding. Notable cases of this kind came under my own observation. In this respect cerebro-spinal meningitis corresponds with other grave epidemic diseases, as for example Asiatic cholera, whose ravages are largely in the crowded and dirty quarters of the poor and degraded.

It is almost the unanimous opinion of physicians who practised in the New York epidemic, that this disease is not at all contagious, or if contagious, it is so only under exceptional conditions, and in a very slight degree. The doctrine of non-contagiousness also receives support from the fact observed in family practice in all epidemics, that cases occur here and there for the most part singly, and often at a considerable distance from each other, and without any inter-communication between patients. Thus, in the epidemic of 1872, I treated thirty-nine cases, occurring singly in families, while I was called to only eight families in which there were two cases, and to only one in which there were three, although free access was allowed to the sick-rooms in most of these families.

Epidemics of cerebro-spinal meningitis are not limited to the human species, as already stated above. The epidemic of 1872 was preceded by one of the same kind among the horses in this city, so that in the latter part of 1871 many perished, having presented very similar symptoms to those in man. The mortality from this cause was especially great among the horses in the large stables of the city car and stage lines. Dr. *Gallop*, the chronicler of the epidemic which occurred in Vermont in 1811, stated that even the foxes seemed to be affected with it, so that multitudes were killed near the residences of the inhabitants. That the lower animals are affected is due to the pervading nature of the cause. Although the New York epidemic occurred among the horses prior to its affecting the people, facts observed confirm the belief that it was not communicated from them, for the stablemen did not seem to be more frequently attacked than others, who were not exposed to these animals.

In a considerable number of instances we are able to discover some exciting cause of the attack, such as bodily fatigue, mental excitement, prolonged abstinence from food, followed by overeating, or the use of indigestible and irritating food. In a case which I observed, a delicate young woman, in charge of one of the departments in a large store, was anxious and overtaxed in the preparation for the annual re-opening, and within a day or two the disease began. In another instance, a boy sickened after a day of unusual exposure and fatigue, having in the mean time bathed in the Hudson when the water was quite cool. In the New York epidemic, those children seemed to me especially liable to be attacked who experienced the severe discipline of the public schools. In one instance in my practice, a girl of ten years returned from one of these schools unusually excited, because she had failed in the annual examination and was not promoted, and in the evening, after she had diligently studied her lessons for some hours, the meningitis commenced with violent headache. The symptoms were severe and characteristic, but she ultimately recovered. During the war of 1812, the epidemic was unusually violent, according to Dr. *Gallop*, among the troops who were fatigued by forced marches, and greatly depressed by a repulse which they had sustained from the British, and during the late civil war, an outbreak of cerebro-spinal meningitis occurred among the soldiers who, according to Dr. *Frothingham*, "were drilled to the full extent of their powers, often to exhaustion."

Statistics show nearly equal liability of boys and girls to this disease, men being more liable than women only as they lead a life of greater excitement, hardship, or irregularity. Children are much more apt to be attacked than adults. In the New York epidemic of 1872, about three-fourths of the cases were under the age of ten years. The older the individual after the period of childhood the less the liability, and after middle life there is nearly an immunity.

SYMPTOMS.

Von Ziemssen in Vol. II. states that there is an abortive form of cerebro-spinal meningitis. Such cases have also been observed in this country, especially in the epidemic of 1872, and also occasional cases which were so mild that the patients were never seriously sick and did not keep the bed. The following were examples of these two forms of the malady observed by myself, and printed in my treatise on *Diseases of Children:* A boy of eight years, previously well, was taken with headache, vomiting, and moderate febrile movement on April 2d, 1872. The evacuations were regular, and no local cause of the attack could be discovered. On the following day the symptoms continued, except the vomiting, but he seemed somewhat better. On April 4th, the febrile movement was more pronounced, and in the afternoon he was drowsy and had a slight convulsion. The forward movement of his head was apparently somewhat restrained. On the 6th, the symptoms had begun to abate and in one week from the commencement of the attack, his health was fully restored.

A boy aged six years was well till the second week in May, 1872, when he became feverish, and complained of headache. At my first visit, May 14th, he still had headache, with a pulse of 112. The pupils responded to light and the right pupil was larger than the left. The bromide and iodide of potassium were prescribed, with moderate counter-irritation behind the ears. The headache and febrile movement soon abated, the equality of the pupils was restored, and in little more than one week from the first symptoms he fully recovered.

On January 4th, 1872, in the midst of the epidemic of cerebro-spinal meningitis, I was called to a girl of thirteen years, who was seized with vomiting, followed by headache, in the last week in December. During a period of six or eight weeks, or till nearly March 1st, she presented the following symptoms: Daily paroxysmal headache, often more severe in the forenoon; neuralgic pain in the left hypochondrium, and sometimes in the epigastric region; pulse and temperature sometimes nearly normal, and at other times accelerated and elevated, both with daily variations; inequality of pupils. This patient was never so ill as to lie in bed during the day, and she usually sat quietly in a chair or on the lounge. Quinine had no appreciable effect on the disease.

There can, I think, be no doubt that these cases pertained to the epidemic, the first two having been abortive cases, and the other so mild that it might not have been recognized except for a few characteristic symptoms.

This disease, as stated by Prof. *von Ziemssen*, ordinarily begins abruptly, but occasionally there is premonition for a few hours or days. In occasional cases I observed premonitions, as chilliness and languor, especially in those who were mildly affected. The ordinary commencement is with a rigor or a feeling of chilliness, or a distinct chill, followed immediately by pronounced symptoms. Sometimes there are two or more chills occurring at irregular intervals. In children eclampsia often takes the place of the chills. Headache, frequently severe so as to extort moans, or profound drowsiness succeeds. Occasionally, in grave cases, the state is one of semi-coma, or coma, which speedily proves fatal. The patient usually lies quiet for a time, and then complains of violent headache, associated with or alternating with, in many cases, equally severe pain in the neck, or in some part of the trunk or in the limbs. The pupils early indicate the nature of the attack by unusual dilatation, or less frequently by contraction, or inequality, or oscillation.

Vomiting occurs early and with little apparent nausea, as in the ordinary meningitis of childhood. It was recorded as an early symptom in 48 out of 61 cases observed by Dr. *Sewall* and myself, while its absence was recorded in only one case, no record having been made of it in the remaining 13 patients. As in diphtheria and scarlet-fever, the temperature is usually at its maximum in the first days. The highest temperature which I observed in any case was in a child of three years; only two or three hours after the beginning of the attack, the thermometer in the axilla indicating 107⅗°. Exceptionally the initial symptoms are mild, and they become gradually more severe, so that a few days elapse before they are so pronounced that the diagnosis is clear. The headache, in some patients chiefly frontal, in others chiefly occipital, continues during the acute stage. Most patients experience also pain elsewhere as in the nucha, along the back, in the anterior aspect of the trunk, or in one of the limbs. It is commonly most persistent and severe in the head or along the spine, but it is apt to shift from one locality to another, so that the patient at one moment complains of headache, and at another of equally severe suffering elsewhere. In cases that progress favorably, the pain begins to diminish in the second week, and by the beginning of the third week is considerably moderated. With the headache, dizziness is common, so that support may be required in attempting to stand or walk. The delirium which certain patients exhibit is commonly of the passive kind. It may resemble alcoholic intoxication. One of my patients, a boy of five years, seemed for the most part rational, but constantly mistook his mother, who nursed him, for another person. At times the delirium is active and violent, so that restraint is required. This form of mental aberration is commonly preceded by intense headache. Certain patients in my practice seemed totally insensible at the time of examination, when it appeared, afterward, that they were conscious of what was said and done.

Restlessness is a common symptom with many on account of the mental state and the pains. Those severely affected ordinarily slumber or are quiet for a few moments, and then roll or throw themselves to another part of the bed. When comatose, or having a mild type of the meningitis, or after the second week, when the acute symptoms have begun to abate, the patient not infrequently lies quiet in bed most of the time. Hyperæsthesia or exaggerated sensitiveness of the surface, due to the spinal disease, is a prominent symptom during the first weeks. Friction upon parts of the surface, or slight pressure, gentle attempts by the nurse to open the eyelids, raising the head, or moving the limbs are resisted, and are evidently painful. Sometimes, in my cases, slowly introducing the thermometer into the rectum caused outcries, from the hypersensitiveness of the anal or rectal surface. Tonic contraction of certain muscles, or groups of muscles, is a noteworthy and prominent symptom, and one of diagnostic value. In a small proportion of cases it is absent or is not pronounced, namely in those in whom the disease affects mainly the encephalon, while the spinal cord is not involved. It is most frequent in the muscles of the nucha, causing retraction of the head, but it is also common in the posterior muscles of the trunk, causing opisthotonos, and also in less degree in the muscles of abdomen and lower extremities, so that patients obtain most relief with thighs and legs flexed. The muscular contraction ordinarily begins at the close of the first or on the second day, and it continues from three to five weeks. In one case, an infant of seven months, it had not abated by the tenth week. Although muscular weakness and unsteady movements of the limbs are common when the meningitis has continued a few days, actual paralysis is unusual, but it sometimes occurs. It may be limited to a limb, or it may be more general. Thus *Wunderlich* relates a case in which there was paraplegia, and Dr. *Law* another case occurring in the Dublin epidemic of 1865, in which all the extremities were paralyzed. Due to the spinal disease, it usually abates during convalescence, but in *Wunderlich's* case there was only partial return of muscular power after five months.

The vomiting which I have alluded to as an early symptom usually ceases after a few hours or days, but is apt to return during periods of recrudescence, which are common in the progress of the malady. It is similar, in its causation and nature, to the vomiting so common in the ordinary sporadic meningitis of childhood. During the intervals of vomiting in the beginning of the malady, a distressing sensation of depression or faintness in the epigastrium is experienced by some. The tongue is covered by a thin, whitish fur, or exceptionally by a dry and brownish fur, as in typhoid and typhus fevers. Anorexia, thirst, and constipation are common. In consequence of the impaired digestion and nutrition, patients progressively lose flesh and strength.

As related by *Von Ziemssen* in regard to European epidemics, the pulse and temperature have been accelerated and elevated in cases observed in this country, and marked daily variations have been noted in both these symptoms. I have records of the temperature in thirty-five of my patients taken before the close of the second week. The lowest was 99½° and the highest 107⅗°. This high temperature occurred on the first day in a child of three years. It fell a little subsequently, but rose again to 107° on the third day when she died. The temperature of the extremities undergoes much greater and more sudden variations without appreciable cause than in any other disease which I now recall to mind, the fluctuations extending over several degrees in the same day, or on successive days. Thus in one of my patients, while the internal temperature varied between 101½° and 104⅕°, the temperature of the hands and fingers on four consecutive visits was 90½°, 90°, 103° and 83°. The sudden and great variations in pulse and in internal and external temperature have diagnostic value in doubtful and obscure cases.

The respiratory apparatus is not notably involved in mild cases, but in severe cases, likely to be fatal, intermittent, sighing, or irregular respiration occurs, but less frequently than in sporadic meningitis. In thirty-one observations in children who had no complication, I found the average respiration forty-two per minute, while the average pulse was 137. It is seen, therefore, that the respiration as compared with the pulse was proportionately more frequent than in health.

The skin in the first days is usually dry. Afterwards perspirations are not unusual, and free perspirations sometimes occur about the head and face. Papilliform elevations due to contraction of the muscular fibres of the corium, the so-called goose-skin, dusky mottling of the surface in grave cases, bluish spots and patches due to extravasation of blood under the skin, minute red points over a considerable extent of surface, and herpetic eruption, chiefly upon parts of the surface which are supplied by branches of the fifth pair of nerves, are common in ordinary cases. Occasionally swelling and tenderness of certain joints occur. Thus, in one of my cases, the parents, who were poor and had endeavored to get along without a physician, supposed the disease was rheumatism, and had been treating it accordingly.

As regards the special senses, the organs of taste, smell and touch are not in most instances seriously involved, but in one case in my practice the sense of smell was entirely lost in one nostril. The eye and ear, however, are frequently the seat of serious lesions, strabismus is common, occurring at any period of the meningitis and lasting a few hours or for days. It may appear and disappear several times before convalescence is established. Although it may continue several weeks, the parallelism is ordinarily at length restored. But a boy whom I saw three months after convalescence still had convergent strabismus of the right eye, and double vision. Changes in the pupils, as dilatation, less frequently contraction, oscillation, inequality, feeble reaction to light, common in the first and second weeks, have been sufficiently alluded to.

Uniformly diffused hyperæmic redness of the conjunctiva is common, not so dusky as in typhus, and with less distinctness of the vessels than in that disease. Now and then there is conjunctivitis, and occasionally it is so intense that the lids swell and are separated by chemosis, while a muco-purulent discharge flows from under them. In the few instances in which I have observed this chemosis, the deeper structures of the eye were also involved, and the type of the disease was severe.

In certain melancholy cases, there is general ophthalmitis, all the important constituents of the eye being involved. The media grow cloudy. The pupils are uneven and obscured by fibrinous exudation, the iris is dimmed and lustreless. Therefore the deeper portions of the eye cannot be readily inspected, but they are seen to be dusky red, when the inflammation is recent, adherent to each other, and infiltrated by inflammatory products. The cornea may ulcerate and burst, with loss of the liquids and shrinking of the eye, but this is unusual. As the inflammation abates and the hyperæmia disappears, the deeper structures of this organ present a dull white color, the lens becomes cataractous, and vision is irreparably lost. The eye atrophies even when there has been no escape of its humors. Prof. *Knapp*, of New York, says: "The nature of the eye affection is a purulent choroiditis, probably metastatic," but as *von Ziemssen* says, it is attributed by many to an extension forward of the inflammation along the optic nerve. Fortunately such severe and destructive inflammation of this organ is infrequent, though hyperæmia of the optic disc and moderate conjunctivitis, which subside without loss of sight, are not unusual. This severe inflammation of the eye is usually bilateral, but in one fatal case which I saw it was unilateral.

Like the eye, the ear may be mildly or severely involved. Otitis media sometimes occurs, presenting a similar history to that in scarlatina, abating with or without perforation of the drum. But there is another malady of the ear, much more grave and destructive to hearing, which appears to be peculiar to cerebro-spinal meningitis, and is not uncommon during epidemics of this disease. Some patients lose their hearing entirely and permanently, and often with fewer subjective symptoms than in otitis media. The loss of hearing does not occur at the same period in all cases. The majority thus affected are permanently deaf when they emerge from the stupor of the meningitis and consciousness returns, but some are not totally deaf at first, but become so during convalescence.

It is a remarkable fact that this loss of hearing is bilateral, and that the deafness is total. I have notes of ten cases of deafness thus produced, in the epidemic of 1872, most of them occurring in my own practice, but one or two were related to me by other physicians. The percentage of this otitis was nearly one in every ten cases. Prof. *Knapp*, who examined thirty-one cases, says: "In all, the deafness was bilateral, and, with two exceptions of faint perception of sound, complete. Among the twenty-nine cases of total deafness, there was only one who seemed to give some evidence of hearing afterwards."

There are two theories relating to the etiology of the deafness, the one ascribing it to inflammatory lesions at the base of the brain, either around the root or in the course of the auditory nerve, before it emerges from the cranium, and the other, to disease of the ear itself. The fact that the deafness is bilateral, and that it occurs simultaneously on the two sides, comports best with the doctrine of a central lesion. Nevertheless the theory that the loss of hearing results from disease of the auditory apparatus, especially of the labyrinth, is more probable, and is, I think, generally accepted by pathologists who have examined ct, and by aurists. Drs. *Keller* and *Lucae*, in three post-mortem :ions, found inflammatory lesions in the labyrinth. *Sanderson*, onograph, alludes to a specimen in the Museum of the *Charité* , removed from the body of a soldier who died of cerebro-spinal is complicated with deafness. In this specimen, "fibrinous adxisted between the bones of the internal ear and the walls of the ." The case of a young woman is also related, in whom the embrane of the semicircular canals was loosened and thickened, anterior canals contained semifluid purulent masses. Moreover *ening*, of this city, obtained the normal reaction of the auditory thin the cranium by electrization in cases of this deafness, by is inferred that the cause of the deafness is extra-cranial. Prof. ays: "The nature of the disease is, in all probability, a purulent ation of the labyrinth."

ANATOMICAL CHARACTERS.

changes which the blood undergoes are due in part to the ional and asthenic nature of the disease, and in part to mmations. As in other forms of inflammations, the fibrin is when the meningitis has continued a few days, and according xtent of the inflammation. Analyses of the blood (*Tourdes*, d *Maillot*) show an amount of fibrin varying from 3.40 to 6 1,000. In the more asthenic and malignant cases which end he blood is found unusually dark and fluid, and containing a few l soft clots. Sometimes bubbles of gas have been observed in vessels and in the cavities of the heart, when only a few hours psed after death, indicating a malignant form of the disease. ations of blood, frequent in grave cases, which occurring in and e skin have given the name spotted fever to this malady, have observed of small extent in and upon the thoracic and aborgans.

ses which are speedily fatal, the brain and meninges are intensely ic, the incised surface of the former presenting numerous vasints, while the cranial sinuses are engorged with dark blood g soft clots. The exudation of fibrin and pus occurs early in es of the pia mater and underneath this membrane over the f the brain. It is most abundant in the fissures and depressions, inter-gyral spaces, along the course of the vessels, upon and he optic commissure, in the space between this commissure and Varolii, and also upon the pons and medulla oblongata. This n had already commenced in certain reported cases which terfatally within twenty-four hours.

inflammation, unlike that in sporadic cases, occurs with nearly quency upon all parts of the cerebral meninges, but, as already seems most intense, or its lesions are most marked, in the dewhere the vascular supply is greatest. The exudation of pus is es in excess of that of fibrin. Thus a case ending fatally in five hours is related in the *Dublin Quart. Journ.* for 1866, in which a purulent greenish exudation had already occurred in places under the meninges.

In cases of great severity, the exudation of fibrin and pus sometimes occurs over nearly every part of the cerebral surface. Thus in an adult negro, who died in Bellevue Hospital in 1872, the post-mortem record states that there was purulent exudation over the entire surface of the cerebrum and cerebellum. The amount of serous exudation also varies greatly in different cases. It may be so small as scarcely to attract attention, or it may be very large. In the *American Journal of Med. Sc.* for October, 1866, a case is related in which three pints of turbid serum escaped from the cranial cavity when the calvarium was raised. A true chronic hydrocephalus sometimes results, producing expansion of the cranial arch. One such case I saw remaining from the epidemic of 1872, in which, one year afterward, the voluminous expansion of the head presented all the appearances of ordinary congenital hydrocephalus.

While, in the first days, there is intense hyperæmia of the brain and meninges, at a later date this gradually disappears. Or if there is much effusion of serum, the convolutions are flattened and the blood-vessels so compressed that there may be less than the normal amount of blood circulating in the brain, and its substance may seem unusually white. Such a case is related by *Burdon-Sanderson.* The patient, a child of three years, died on the sixteenth day, and its brain was everywhere anæmic, while the ventricles were greatly distended by turbid serum. As in sporadic meningitis, there is ordinarily more or less serous effusion in the lateral ventricles.

Softening of the brain is common. At one of the necropsies in Charity Hospital, on Blackwell's Island, N. Y., in 1872, made only seven hours after death, in a case that terminated in three days, the brain generally was softer than in the normal state. In one of the Bellevue Hospital examinations, the fornix, corpus callosum, and septum lucidum were softened, and in another case the cerebral substance adjacent to the subarachnoid space had undergone similar change. Localized cerebral softening has been recorded by various physicians as of the medulla oblongata, pons Varolii, and portions of the hemispheres by Dr. *Moorman,* and the upper part of the left cerebral hemisphere by Dr. *Upham.* Besides, ordinarily, as in sporadic meningitis, the walls of the lateral ventricles are softened. Œdema of the entire brain occurred in a case published by Dr. *Hutchison,* in the *Amer. Jour. of Med. Sci.* for July, 1866. The patient was only four days sick, and the brain was so œdematous that serum escaped from the incised surface.

The anatomical changes which occur in the spinal cord and its covering are similar to those in the encephalon. The intense hyperæmia of the meninges is soon followed by fibrinous, purulent, and serous exudation in and under the pia mater. In severe cases, the entire spinal pia mater is infiltrated with inflammatory products, but in other cases, and usually in those of a mild form, a portion only of this membrane is involved, more frequently the posterior than anterior. The exudation, which consists of serum, pus, and fibrin in varying proportion, and is ordinarily white or greenish, is sometimes blood-stained from extravasations. In a case related by *Burdon-Sanderson*, there was a purulent and fibrinous layer one-eighth of an inch thick, over the entire cord below the bronchial swelling. The cord itself is in some patients hyperæmic, and in others of nearly normal vascularity, while occasional softening of portions has been observed.

Other lesions have been recorded in the organs of the thorax and abdomen, in a certain proportion of cases, as pulmonary œdema, nodules of hepatization, and small extravasations. Serous transudations, sometimes blood-stained, occasionally, occur in the pleural and other serous cavities. The cavities of the heart contain more or less blood, in which are dark and soft clots in the malignant and speedily fatal cases, and larger and firmer clots in cases which have been more protracted. Congestion of one or more of the organs of the trunk has been noticed in some cases while in others their condition was normal. The spleen, which is so often enlarged and softened in infectious maladies, presents in certain cases no appreciable change, while in others it is somewhat congested and swollen. Unusual prominence of the solitary and agminate glands has also been noticed, but not uniformly.

TREATMENT.

Preventive.—Observations in the various epidemics show that this disease occurs most frequently, and assumes its severest and most fatal type, where sanitary requirements are most neglected. Among the poor, living in the filthy streets and crowded tenement houses of the city, a much larger proportion are victims than in the better walks of life. The New York epidemic of 1872 afforded striking exemplification of this fact. Therefore, by proper sewerage and drainage, by cleanliness, personal and domiciliary, and the removal of all refuse and decaying matter from the streets, cellars and yards, so as to procure the utmost purity of the air and the use of plain, easily digested, and wholesome food, the danger of contracting the meningitis during an epidemic is greatly diminished. Of great importance also, in the way of prevention, is the avoidance, during an epidemic, of all irregularities in the mode of life, of mental and bodily fatigue, of harassing cares and depressing emotions, since these act as exciting causes, as the history of many cases shows. A quiet and regular mode of life, with full amount of sleep, and regular meals of plain, nutritious, and easily digested food should be advised, especially for children and young people.

Curative.—The anatomical characters ascertained by many post-mortem examinations suggest the treatment, which indeed experience has found to be most useful. The intense inflammatory hyperæmia of the cerebro-spinal axis, which is developed as soon as the first symptoms occur, demands prompt treatment. It is best relieved by the application of bags or bladders of ice over the head and to the nucha.

A hot mustard foot-bath or a general tepid bath is also useful, as it produces a derivative action from the nervous centres, and tends to diminish the excited and sensitive state of the nervous system and prevent convulsions.

The abstraction of blood as a means of relieving the cerebral hyperæmia was commonly employed until within the last twenty-five years, but it is now for the most part abandoned, on account of the asthenic nature of the disease. Venesection is entirely discarded, but some intelligent observers, as *Burdon-Sanderson* and *Niemeyer*, have in recent times advised the sparing abstraction of blood from the head by leeches in the commencement of the more sthenic cases, when symptoms of impending coma were present. But it is surprising to observe how the profound stupor will pass off in a large proportion of cases without loss of blood, by the application of ice to the head, and with other treatment recognized as appropriate; and the danger in the subsequent course of the case is seen to be in great part from the prostration, which the loss of blood would have augmented. In the epidemic of 1872 I saw cases apparently hopelessly comatose in the first hours of the malady, and some of them were pronounced by competent physicians to be beyond the reach of remedies, and yet after two or three days they became more conscious and either recovered by degrees, or died days or weeks subsequently, not from coma as much as from prostration or softening in the nervous centres. In only one of the cases which I treated in the epidemic of 1872, did I recommend leeching. The patient, a girl of four years, was in convulsions which ushered in the malady, and insensible, and only a moderate amount of blood was removed from the temples, but I had occasion to regret this treatment, for she died extremely wasted and feeble nine weeks afterwards. She required, long before the fatal termination, all the strength which could be imparted by the most nutritious diet and the liberal use of alcoholic stimulants.

The employment of the bromides is indicated, in ordinary cases, in order to diminish the intense cerebral hyperæmia, allay the excitement of the nervous system, and prevent convulsions. They should be given in decided doses as soon as the symptoms indicate the nature of the disease. In the New York epidemic, we commonly prescribed the bromide of potassium in five or six grain doses, every second hour to a child of five years, but more frequently if convulsions occurred or were imminent. It can be given in frequent and large doses for a few days without ill effect; but its long-continued use, unless there are clear indications for it, is to be deprecated, since it produces now and then, when employed for many days, symptoms (bromism) which can with difficulty be discriminated from those of cerebro-spinal meningitis, such as muscular weakness, dilated pupils with perhaps impaired vision, unsteady gait, nausea or vomiting, with abdominal pain. Frequent and large doses should as a rule be prescribed only in the first week, after which this remedy should be discontinued entirely or given sparingly, but its use may be resumed from time to time, during periods of recrudescence, which are very apt to occur.

The intense headache and consequent restlessness which characterize many cases require, in addition to the bromide, either the hydrate of chloral or an opiate. An opiate is, I think, in most instances preferable, and a moderate dose suffices. A patient of six years, in my practice, was quieted by one thirty-second of a grain of sulphate of morphia.

Another remedy scarcely less useful than the bromide is ergot, from its known effect in contracting arterioles, and diminishing the arterial supply to the cerebro-spinal axis. It can be administered in the tincture, fluid extract, or wine. The alkaloid, ergotin, is sometimes employed in pill or solution, or given hypodermically in water, with a little glycerin. I prescribed a one-grain pill of ergotin to be taken every six hours to a child of 13 years. The efficacy of ergot is most marked during the first or second week, when the congestion of the nervous centres is greatest. At a more advanced stage, when there is less congestion and the danger arises more from the inflammatory products and structural changes, as softening, the time for the use of ergot is past, or if still of some service, it is less urgently required than at first.

The similarity of the lesions to those in sporadic meningitis in the treatment of which iodide of potassium is in common use, suggests the employment of this agent. It probably aids in the removal of the liquid portion of the exudation. I have prescribed it in combination with the bromide, and alone when the bromide was suspended.

Quinia does not seem to exert any marked controlling effect either on the course of the disease or the pains, although the severe pains are apt to be paroxysmal, so as to indicate the need of this agent as an anti-periodic. I have employed it in large and small doses, in one instance giving fifteen grains daily to a child of thirteen years, but do not know that I have derived any benefit from it except as a tonic.

Sustaining measures are indicated from the first. The diet must be nutritious during the entire course of the malady, consisting of the animal broths, milk, etc. After the violent initial symptoms have abated, alcoholic stimulants are needed, and they should be prescribed in all cases, however early, in which the pulse is feeble, and there are evidences of marked prostration. When the danger from the intense cerebro-spinal hyperæmia has been averted, tonics, especially the ferruginous, may also be employed to aid in arresting the profound blood changes. Laxative enemata should be prescribed to relieve constipation, and rectal alimentation should be resorted to in those cases in which frequent vomiting prevents proper nutrition in the natural way. Dry cupping should be employed along the spine two or three times daily, or if for any reason the use of the cups is not satisfactory, a stimulating embrocation, as that of equal parts of turpentine and camphorated oil, should be prescribed. Visitors should be excluded, and the room should be dark and quiet, for anything that annoys and excites the patient, whether loud noises or talking, or a bright light, or the use of indigestible food, has, in my opinion, a tendency to aggravate the malady.

SYPHILIS.

BY

JAMES NEVINS HYDE, M.D.,

OF CHICAGO,

PROFESSOR OF SKIN AND VENEREAL DISEASES, RUSH MEDICAL COLLEGE, CHICAGO.

SYPHILIS.

NOTE.—Professor HYDE has supplied the Editor with a very voluminous and carefully selected bibliography containing nearly five hundred references. It is with great regret that the Editor feels obliged to omit it; but the space at his disposal for the subject of SYPHILIS being limited (as for each other subject), he has concluded, with the kind consent of the writer, to omit the bibliography rather than abridge the very valuable article prepared by Professor HYDE.—EDITOR.

In the following pages it is intended, as far as possible, to refer briefly to those facts in the diagnosis, pathology, and treatment of syphilis and the chancroid ulcer, which have been in various degrees illuminated by investigations made since the first edition of *v. Ziemssen's* Cyclopædia was issued from the press.

The progressive relative infrequency of the chancroid ulcer ("simple," "soft," "non-infecting" chancre), as compared with the initial lesion of syphilis, has attracted the special attention of French authors, whose observations are based upon statistics. In this country a similar conclusion is chiefly derived from the personal experience of practitioners. It is probably true that, in the course of extensive wars, when large bodies of young men are removed from their usual domestic and social surroundings, the crapulous chancroid multiplies, not only when regiments of men have special opportunities for propagating it, but also for a time after the troops return to their homes in periods of peace. Thereafter, gradually decreasing in frequency, the non-infecting sore is relatively replaced by the initial lesion of syphilis, the latter increasingly observed as the social conditions of the infected improve.

In the site of a chancroid ulcer, the following pathological conditions are observed: The floor of the lesion is constituted of a uniform, deep cell-infiltration, terminating abruptly below, and extending laterally beyond the visible margins of the sore. The tissues next this infiltrated mass, exhibit loose meshes and occasional cells with large nuclei. Thickened and infiltrated papillæ are visible in the swollen peripheral border, and the mucous layer dipping down between them is correspondingly thickened. But a cell-infiltrated corium is exposed at the level of the floor of the lesion, where no papillæ are longer visible. In the infiltrated mass beneath, there is a close network of faintly contoured, broad and narrow fibres with many nucleated cells distributed among them, some as large as lymph-corpuscles, others smaller. The capillaries are enlarged from thickening of wall and increase in size of lumen. This is evident both in the infiltrated and contiguous œdematous structure. Degeneration of all elements occurs in the upper portion of the infiltration, and is proportioned to the extent of the latter.

Extra-genital sores of this character and those occurring upon the cervix uteri are rarely noted.

Chancroids have been satisfactorily destroyed by the aid of a platinum wire brought to a white heat with a galvano-caustic apparatus, a revival of a method formerly advocated. Their local treatment by some of the stronger mercurial preparations has been followed by disagreeable and even dangerous sequelæ. The employment of iodoform, however, for a similar purpose, has steadily increased in favor. The chief objection to its use is the penetrating, peculiar, and disagreeable odor which it diffuses about the person. This is in part remedied by its admixture with some of the essential oils, while the energy of its action is reduced by the addition of tannin or Fuller's earth. It not only hastens the process of repair, but produces a local anæsthetic effect.

In the treatment of phagedenic chancroids, brilliant results have been obtained by long-continued immersion in water of the temperature of 98° F. The patient rests upon a comfortable seat in the bath-tub, and either continues there night and day until relieved, only leaving the water for the purpose of emptying the bowel and bladder, or from ten to twelve hours of continued immersion in the day are succeeded at night by other appropriate local measures, till the process of repair is completely assured. The treatment is accompanied by great relief of pain.

Referring to the subject proper of this review, it is needful to admit at the outset that the exact nature of the disease syphilis, and of what is best recognized as "the syphilitic virus," is yet unknown. It has been assumed that syphilitic inoculation is effected by contact of leucocytes with a degenerate amœboid corpuscle or disease germ, and that this self-multiplying germ is gradually conveyed to the blood-current through lymph spaces and channels. Rapid proliferation follows, till the lymphatic circulation is impeded by a deposit of arrested normal material. According to the latest hypothesis, the initial sclerosis is merely a point of special irritation, this irritation being conveyed to the nervous centres and reflected thence to the skin and mucous membranes in a particular series of phenomena. Mercury, which excites an antagonistic series of actions, proves in a similar manner its therapeutic value. These and other like theories have served merely to attract renewed attention to a question of profound interest.

The initial lesion of syphilis, usually solitary, is not rarely multiple at the outset. In seventeen individuals examined by *Rizot*, there were ninety-two chancres in all, sixteen upon the person of one man. It is an error, however, to conclude, with this author, that multiplicity is the rule. Nor should it be forgotten that an extraordinary multiplicity of lesions would have no weight upon either side of the question respecting prognosis and gravity of future symptoms.

Follicular chancres of the vulva in adult females are dry hypertrophic lesions, located upon the external face of the labia majora, and usually multiple, more or less dense and flat, pin-head-sized papules, slightly umbilicated at the apex. Histologically they are rather peri-follicular than follicular, and should, according to *Fournier*, be thus appropriately named. One or more hairs can generally be distinguished projecting from the centre of each papule, which furnishes a scanty secretion. These infecting lesions are too readily confounded with simple so-called follicular vulvitis.

Genital chancres of infants infected by improper contacts are interesting in a medico-legal point of view. Vulvar lesions, rare elsewhere, are here the rule. They are most commonly seen on the inner face of one labium majus, near the clitoris, "the superior and most projecting portion of the vulvar canal," and are accompanied by rapid development of constitutional disease.

Multiple herpetiform chancres are not to be mistaken for the simpler lesions of herpes progenitalis. They are shining, dark-red, circular, hæmorrhagic excoriations, numbering from five to fourteen upon a single individual, not accompanied by the itching and burning sensations induced by the non-infecting herpetic disease, and distinguished naturally by characteristic sclerosis and adenopathy of the groins. Another exceptional form of infecting primary lesion is the diphtheroid of the glans, where the part named is covered with an indolent, glistening, grayish-tinted, leathery, elevated hood, without underlying erosion, its edges continuous with the peripheral normal membrane, and destitute of induration. The infecting forms of balano-posthitis generally display a reddened, thickened, and partially or generally excoriated præputial membrane, which thus serves to distinguish the first symptom of syphilis. In all these exceptional forms, the characteristic induration may be altogether absent, or present but for relatively brief periods, or present in the scarcely noticeable varieties of the "parchment" form. These peculiarities they share with most initial lesions of women which are genital and intra-vulvar.

The initial sclerosis, examined histologically, exhibits a cell-deposit in the papillæ and throughout the entire thickness of the corium to the subcutaneous tissue, limited abruptly at the sides and below, and surrounded by a coarse tissue of fibres containing irregularly distributed cells with large nuclei. The infiltrated cells of the indurated tissue are roundish, with one or two nuclei and a finely dotted protoplasm overlying the inclosed nucleus. The narrow-meshed enveloping network and its cell-deposit are irregularly exposed at the surface of the ulcer, whose secretion is seen to contain nuclei and nucleoli with small and large shrivelled cells filled with granular elements. Thickened, club-shaped, and cell-infiltrated papillæ, with an atrophied rete running between and over them, appear on the border of the exposed ulcer. The vascular elements are represented in thin-walled capillaries of diminished lumen; this change being probably an effect and not the cause of the sclerosis. The distinguishing feature of this initial sclerosis of syphilis, when contrasted with the chancroid ulcer, is the tendency of the former to hyperplasia, to a peculiar species of new-growth, which in the latter is replaced by a distinct retrograde metamorphosis, the process of destruction being inaugurated at the earliest appearance of the sore.

The sclerosis of the initial lesion of syphilis, resulting from hypertrophy of connective tissue in the cutis, is said to be composed of pure collagen. When this involves the adventitia of the vascular elements, it usually affects also the connective tissue surrounding the sclerosed vessel, and the lymph meshes disappear. The vasa vasorum are early implicated and, when these do not exist, the obliterating endarteritis begins in the outer coat and not in the endothelium. A form of granulating endarteritis has also been observed where the vessels are obliterated by deposits and accumulations of round cells. But both processes may continue, or the one may supplant the other. The larger vessels are usually obliterated by the first, the smaller by the last-named method.

The results of the recently revived operation for the excision of the initial sclerosis, carried even to the point of præputial circumcision and removal of the labia, with a view tó prevent the occurrence of constitutional infection, are not encouraging. The operation has gained special favor only with those who practically deny that there are venereal sores in no way connected with syphilis; and the suspicion attaches to the brilliant results reported after excision, that syphilis might not have followed if the lesions had been unmolested. The amenability of the initial sclerosis, often an insignificant symptom clinically, to mild constitutional and local treatment, is a bar to operative procedures of severity which do not furnish a guarantee of immunity.

Constitutional syphilis consummated, the proof is tolerably clear that all the physiological secretions of the infected individual (more particularly the milk, tears, saliva, and semen), are incapable of communicating the disease to a healthy person. In the reported cases of infection by these vehicles, it has generally been discovered that they were commingled with pathological fluids or products of the syphilitic process, such as secretions from labial mucous patches. Reports of instances of mediate contagion have lately multiplied where domestic utensils, toothbrushes, cigars, toys used in the mouth, and the instruments employed in tattooing have all been cited as disease-carriers. In the process of vaccination it now seems probable that not merely the intoxicated blood of the vaccinifer, but also pathological secretions from ulcers underlying the vaccine vesicle and even epithelial bodies and leucocytes may prove perilous when transferred from the diseased arm in connection with an apparently normal lymph. It has been erroneously claimed, however, that an apparently normal lymph cannot be obtained from the arm of an affected infant.

Experiments in blood-counting by the aid of the hématimètre, when undertaken in the early stages of syphilis, demonstrate that at these periods the red blood-corpuscles diminish in number from one-seventh to one-half, while the white corpuscles, on the contrary, with the quantity of albumen, increase. Thus the chloro-anæmia of these stages is explained. The normal proportion of these elements is restored under the use of mercury and the iodide of potassium when the latter are employed in doses properly proportioned to the requirements of each case.

Beside the glands which are usually affected with adenopathy as a coincidence with the outset and explosion of syphilis, other groups of deep lymphatic ganglia are known to become secondarily engorged, usually in connection with, occasionally without, visceral involvement. Thus the prævertebral, lumbar, iliac, mesenteric, and femoral glands may become the seat of hyperplasia resulting in cheesy, friable degeneration of the substance of the gland itself, or in thickening of the outlying connective tissue. In the phases of tertiary disease, both a sclerous and gummy form of adenopathy have been recognized.

Viewed in relation to other diseases, syphilis, in its general manifestations, is shown by recent investigation to remain distinct and separate in its identity from all. Though termed by an author an "imitatôr" of the processes of most other maladies, it is clear that this resemblance is due rather to the fact that all the tissues and structures of the body obey, in their departures from a normal standard, the same general pathological laws. It is more philosophical to regard syphilis, not as imitating other diseases, but as imprinting its special features upon those recognized as typical of other disorders. There is no evidence that syphilis confuses itself with tuberculosis, lupus, or gout. There is no good support for the asserted doctrine that syphilis, in one or several generations, exhibits symptoms which cannot be recognized as distinct from struma, scrofulosis, etc. In coincidence with several acute and chronic maladies, syphilis appears merely to act as one of other depressing agencies. The same is true, to a certain extent, in cases of traumatism. But here a wound may be transformed into an ulcerative syphilide or a gumma; or its cicatrization may be arrested or impeded by constitutional impairment. A fracture in a syphilitic patient may fail to unite till the proper treatment of the general condition has been adopted.

Respecting the prognosis of syphilis, it is certain that neither from the character or persistence of the primary lesion, nor from the duration of the two periods of incubation, nor yet from the first manifestations of general syphilis, can a clue be obtained as to the future benignancy or malignancy of the attack. It is, however, equally certain that a just distinction can be established between benignant and malignant symptoms actually under observation; that syphilis has a distinct tendency to self-limitation; and that many cases unrecognized and untreated are well-nigh destitute of pathological interest.

The nomenclature of syphilitic cutaneous manifestations has been improved by substituting for such expressions as "syphilitic lichen," "syphilitic psoriasis," etc., the terminology of the papular syphilide, the squamous syphilide, etc. Among these manifestations, undescribed in the former edition of this work, should be named the pigmentary syphilide which is merely a superficial mottling of the surface of the skin in patches, of a dirty-brown to a blackish hue, forming a species of network inclosing areas of natural or unnaturally white integument. The whole is probably an undue pigmentation of the ordinary syphilitic macule or papule, whose removal occurs by absorption, while hyper-pigmentation proceeds in the intermacular spaces. The symptom is most often distinguished upon the neck, shoulders, and bosoms of blonde women, particularly those of a lymphatic temperament, though it is seen also in the individuals belonging to dark-skinned races. Dirt and neglect are its immediate factors.

Gummata of the skin, as distinguished from the classical subcutaneous lesions, are recognized in the form of diffuse infiltrations (showing as raised, reddened, smooth, or slightly scaly patches), and also in distinct localizations which exhibit cutaneous symptoms proper. Such, without question, are the tubercles of the skin appearing in tertiary syphilis (illustrations of which can be seen in most atlases of syphilitic diseases of the skin and mucous membranes), the tubercles either developing into hyperplastic nodules or undergoing destructive metamorphosis, breaking down with characteristic detritus and ulcer-formation.

Turning to the mucous membranes, we there recognize, not merely the well-known erythematous and mucous patches, but, as occurring in late syphilis, scaly patches (throat, tongue, inner aspect of lips and cheeks), with flat, thickened, whitish and bluish epithelial plates of ichthyotic aspect; and also gummatous ulcers of the mucous membrane whose course and ultimate termination in disease of underlying or contiguous bone is suggestive of the same process upon the skin. The distinction between scaly patches of the tongue in syphilis, ichthyosis linguæ, "psoriasis of the tongue," "plâque de fumeurs," etc., is still involved in obscurity. They are not all necessarily of syphilitic origin, though often infection seems to furnish the immediate or exciting conditions of their appearance. They are particularly rebellious to specific treatment. Diffuse and circumscribed varieties of superficial and deep sclerosis affecting the mucous, submucous, and even the muscular tissues of the tongue furnish lamellated indurations which constitute, in one stage, hypertrophy of the tongue, and, in a subsequent period, may be succeeded by atrophy.

Less frequently involved than either the mouth or pharynx, the epiglottis is more frequently the seat of syphilis than is generally thought to be the case, for painful deglutition may be assigned to another cause, and circumscribed gummata or infiltrations in this part may long remain unnoticed. The disease usually appears in from three to six years after infection, in which case the epiglottic lesion, especially if there be much tumefaction and ulceration, may be confounded with cancerous disease. But concurrent symptoms and the history of the case will, in most instances, furnish a clue to the diagnosis, while in tuberculosis of the epiglottis, the granulations are flatter, the tumefaction less considerable, and the signs of pulmonary complication tolerably conclusive. In syphilis of the epiglottis, it should not be forgotten that the emaciation, anæmia, and cough with expectoration, may lead to error.

The bisulphate of carbon has been used with success in the local treatment of the syphilides, but there are obvious objections to its employment. For the relief of painful palmar and plantar lesions, especially those which are persistent and rebellious to treatment, exhibiting much infiltration of the skin and scaling with ulcerated fissures, immersion in hot water is desirable. The water is usually rendered alkaline by dissolution in it of the bicarbonate or the biborate of soda, and somewhat demulcent by the addition of bran. Mercurial oinments are subsequently used with advantage, and in the preparation of these the articles known as vaseline, cosmoline, etc., products obtained by the purification of crude petroleum, are of recognized value and increasingly employed.

A rare symptom of early syphilis is a spontaneous exudation of blood into the skin. This exanthem occurs in discrete and confluent patches, from a millimetre to a centimetre in diameter, the irregularly pigmented sequelæ of which much resemble the petechial blotches in scurvy. Coincident symptoms are arthritic and peri-articular localized effusions of blood. These bleedings are analogous to those described as occurring from the navel of new-born syphilitic infants, and, like the latter, are scarcely to be considered as proper symptoms of the disease, but as originating in individuals otherwise disposed to hæmophilia, who have been infected with the disease.

A tertiary involvement of the sublingual gland has been noted by *Fournier* in one instance, and has been twice observed by ourselves (unreported cases). In all three, rapid disappearance of the tumor followed appropriate treatment.

Syphilis of the rectum is produced by ulcerative or gummatous lesions, the former both within and without the verge of the anus, and may finally beget the non-ulcerative fibro-plastic cylinder of the gut, known as the "ano-rectal syphiloma." It consists essentially of an infiltration into the walls of the rectum and anus. Stricture results from contraction, adhesion, or agglutination of the walls, distinguished, subjectively, by negative symptoms of painlessness and chronicity; objectively by coarctation, abnormal folds and pockets of rectal membrane, impermeability by sound, and fecal accumulations above the site of the obstruction. The entire process is not unlike that which eventuates in laryngeal stenosis. In the latter case there is pain, dysphagia, dyspnœa, and various degrees of aphonia, resulting from degenerative and destructive processes. Stenosis occurs in consequence of œdema, vegetations, membranoid occlusion, and the contraction of cicatrices. . Membranoid occlusion usually first affects the anterior commissure, leaving a posterior opening, though the reverse has been noted.

In a few cases, usually of mild grade, but occasionally severe, medical treatment alone (potassium iodide in large doses) has effected complete relief in both laryngeal and rectal stenosis. But in the treatment of most cases of this character, including syphilitic stricture of the œsophagus, dilatation is requisite by the aid of graduated cylindrical bougies used continuously (where admissible) or interruptedly, covered with flannel smeared with mercurial or other ointment. The galvano-caustic apparatus or the Paquelin cautery may be employed where destructive effects are desirable. Gummy tumors of the larynx, more frequently recognized now than heretofore, occur in diffuse and circumscribed forms, of gray or yellowish tint, single and large or small, multiple and limited to the mucous or submucous tissue. They usually degenerate into ulcers involving the epiglottis, arytenoid cartilages, and other parts. When the deeper tissues are invaded, there is generally a painful perichondritis, accompanied by crepitation on palpation and final caries in advanced cases. Syphilitic paralysis of the vocal chords is usually monolateral and left-sided, and, though not remediless, is rebellious to treatment.

In syphiloma of the heart, a coarse, dense fibrous stroma is found infiltrated with an abundance of small cells. The most common seat of gummata is the wall of the left ventricle. There are usually cerebral complications, and the issue is fatal in about two-thirds of all cases.

Syphilis deposits, especially at the base and middle and lower lobes of the lungs, fibroid masses with granulative tissue, or the neoplasm may exist in the form of bands. Gummy nodules readily degenerate, and their vascular channels are the sources of the hemorrhage. The cheesy degeneration of ordinary pulmonary tuberculosis differs from the necrosis of the small cell-infiltration in syphilitic phthisis. Microscopically the syphilitic lesion shows thickening of the bronchial septa and vesicular coats, with bronchial dilatation. The fibrous septa are crowded with cells and nuclei projecting into the lung-tissue between distended alveolar walls. Subsequently the latter contract and become obliterated, leaving a fibro-nucleated tissue containing moderate-sized vessels. In patches, central softening and cavity formation may be discerned. Thickening of the outer arterial coats (and probably also of the inner) bears no proportion to the general thickening of the bronchial septa and the tissues contained in them. The more chronic forms of tubercular phthisis, "chronic pneumonia," "miners' phthisis," etc., are but varying forms of pulmonary inflammation, and from these the syphilitic forms differ but in their less even distribution, the circumscribed rather than the disseminated forms of their lesions, their symmetry (they are rarely monolateral), the absence of tubular dilatation, and their solidity, which is in general due to a mere abundant cell-proliferation. Looking not unlike red or gray hepatization, many diseased patches in syphilis are merely tougher, less granular, and somewhat more translucent. Gummata of the lungs, usually superficial and limited to the lower lobes, rarely exceed in number six to eight, and when they degenerate centrally, leave walls which are white, hard, and fibrous. Clinically the symptoms are moderate cough and dyspnœa, scanty expectoration, and unless the lesion is circumscribed and superficial, few physical signs. Occasionally the hæmoptysis is severe and abundant. The prognosis, dependent upon the extent of the disease, and the amenability of the patient to treatment, is usually favorable. That the treatment has been effective in procuring relief is demonstrable by the rapidity of amelioration of symptoms under the employment of remedies which would probably aggravate phthisical states, and by post-mortem as well as clinical evidence. However grave, therefore, and extensive the lesion, a fortunate issue may be expected. We have seen the severest hemorrhages with prostration succeeded by a condition of apparently perfect health within six months.

Among visceral complications of syphilis should be named splenic enlargement of adults in acquired forms. Though occurring at any time during the secondary stage, it may occur between five and twelve weeks after infection.

Partial indurations of the corpora cavernosa, merely mentioned in the preceding edition of this work, have been more definitely described as millet-seed-sized, indolent and painless localizations, of insidious beginning, with loss of permeability of the erectile tissue. Thus the corpus of one side only may become turgid when erection occurs, a lateral curvature of the organ resulting, its extremity pointing to the fold of one groin. Similar deposits of syphilitic tubercle are described as occurring near the frenum at the base of the glans; and are not to be confounded with primary venereal lesions, or the peri-urethral phlegmon, which is similarly located, usually to be recognized by a history of blennorrhagia.

Functional impotence in the male, independent of cachexia and lesions of the testis, may occur in the course of syphilis, in consequence of some obscure changes in the nervous centres. In these cases, the external genitalia are ordinarily exsanguinated. When the testis is involved, its secreting structure is replaced by syphilitic neoplasm, incapable of furnishing the male element. Thus may result sterility without impotence.

The subject of nervous syphilis has been fairly brought up to date by *Heubner* in his valuable chapter on this subject, in Vol. XII. of the American edition of this work, to which we refer our readers. It remains to be added merely that *Schott* has, since that chapter was written, more accurately described and illustrated the lesions of the optic nerve, where both conditions of neuritis and perineuritis have been recognized. There is free proliferation of young, round nucleated cells in the connective-tissue sheath, with an increase of spindle-shaped cells. Similar bodies, solitary and in rows, have been discerned in the nervous tissue itself and around the nutrient vessels, the pressure separating and thinning the nerve-bundles. In one case, the process was limited to a portion of one optic nerve, and was more pronounced near its origin. In another case, both nerves were involved, the left more decidedly.

Syphilitic pseudo-general paralysis, accompanied by cerebral exaltation, incoherence of ideas, and hilarity alternating with fits of depression and mania, is to be distinguished from the general paralysis of the insane. The former is preceded by severe cephalalgiæ with nocturnal exacerbations, and is characterized by a less persistent maniacal excitement, occasional complications in the form of curiously selected nerve paralyses, hemiplegia, paraplegia, greater frequency of optic and aural disturbances, anæsthesia, vertigo and contractions, aphasic disorders without facial or labial tremor, cerebral or spinal meningitis, or pachymeningitis, capricious association, succession and disappearance of motor and sensory phenomena, with absence of general progressive muscular paresis, and in the effects of specific medication. There is usually, also, a history of syphilis.

In the diffuse form of muscular syphilis, there is usually pain of a neuralgic character, with more or less rapid contraction of the invaded tissue, resulting in fixation, without a history of preceding rheumatism. The disease occurs in from ten to fifteen months after infection, in both mild and grave forms of syphilis, and is persistent under appropriate treatment. The coincidence is more frequently with non-ulcerative rather than with ulcerative symptoms in the skin. By *Mauriac* the lesion is described as a subacute myositis, with remissions and relapses, terminating occasionally in spontaneous restoration. In congestive myositis, the lower end of the biceps cubiti is most often involved, between six and ten months after infection, in individuals who are suffering from benign forms of the disease, and who have previously complained of vague muscular pains. Pseudo-anchylosis of the elbow results from implication of the biceps brachialis. The lesion is probably hyperæmic in character.

The orange-sized muscular tumors (gummata) which have been described as undergoing suppurative inflammation, are now known not to terminate in this way, an important point in the matter of diagnosis. They may, however, undergo fatty degeneration. Of various shapes, they at times become glued to a pathologically altered aponeurosis, or, occurring at the limit of muscular fibres, affect the tendinous expansion. They are more readily recognized when the muscle is relaxed, and are then painless. The diagnosis is made clear by concomitant symptoms. They disappear under treatment with satisfactory rapidity.

"Tertiary syphilitic housemaid's knee" (*Keyes*) is the most common of the rare forms of bursitis in lues. The bursæ at the inner side of the knee and behind the olecranon are next in order of liability. First appears a deep tuberculo-squamous, possibly ulcerative syphilide, with livid, roughened, elephantiasic and corroded surface, where deep lateral furrows form in consequence of the joint-motion. Before or after the height of the morbid process in the skin is attained, the walls of the underlying bursa thicken, become invaded with gummatous infiltration which finally involves the entire structure and cavity. Gelatiniform softening of the newly formed tissue results in ulceration and discharge of pus and débris. Occasionally shorter or longer lived fistulæ result; cicatrization, however, in cases, may proceed without this annoyance.

In a second, more insidious and painless form, the gummatous infiltration-process extends from within; and the peripheral and integumentary structures sooner or later suffer secondarily. In such cases, a diagnosis is made with greater difficulty. A history of lues, of course, will aid ; and, before the stage of softening, a characteristic, uneven, wooden hardness of the tumor is recognizable.

Macular and papular syphilides of the ocular conjunctiva usually coexist with generalized eruptions of similar type. Discolorations are here usually well-defined, sometimes slightly elevated, and are scarcely more vascular than the outlying tissues, with little proneness to ulceration. Chancres of the semilunar fold, the result of contact with infectious matters, are indolent, indurated, and apt to be of purplish tint. They require differentiation from early lupus of the lid. Ulcerations of the conjunctiva from breaking down of gummata and tubercles have been recognized near the corneal margin, with jelly-like, semi-translucent tissue around the erosions. In Dr. *Bull's* case, a growth surrounded the corneæ of both eyes, and was most distinct externally and inferiorly, elevated to the extent of one or two lines, and situated in and beneath the ocular conjunctivæ. It extended away from the corneæ in every direction to the extent of about one-third of an inch, was non-vascular, knobby, moderately indurated, and yellowish in color. It was glued to the conjunctivæ, and was set in the latter as a watch-crystal is encased in its frame. It cut like brawn, and but slight hæmorrhage resulted. Between the tendons of the superior and external recti muscles, there was extensive gummatous infiltration of the sclera, painful and tender, extending backward symmetrically. The media of the eyes and their deeper tissues were unaffected.

In spite of adverse opinions lately offered, chiefly by writers on the continent of Europe, the views of *Hutchinson* have become even more generally accepted, respecting interstitial keratitis and the notched, pegged, and pointed permanent incisors of hereditary syphilis. Increased clinical experience has placed these observations upon the basis of ascertained fact. In the punctate forms of keratitis, the cornea is covered with disseminated pin-point-sized dots, whitish-gray in hue, not tending to coalescence, and unaccompanied by lachrymation and pain.

Superficial parenchymatous and gummatous affections of the sclera exhibit limited points, hyperæmic at the outset, or split-pea-sized patches and peri-corneal zones, of violaceous tint, enlarging in all directions, and usually productive of but few subjective symptoms. The conjunctiva is, and is not, involved, according to the severity of the process. In extreme cases the cornea becomes opaque, the iris adherent, and cyclitis sets in. Gummata may break down and leave ragged-edged secreting ulcers. The infantile iritis of inherited disease is rare; and usually occurs in children about the fifth month. Often symmetrical, but occasionally confined to one eye, it is rarely as complicated and severe as the acquired affection in the adult. Corneal haziness, pain, photophobia and sclerotic injection are rare. But though the symptoms of acute inflammation seem to be wanting, there is usually abundant lymph exudation and great danger of pupillary occlusion. The disease occurs in those previously treated with mercury, though this remedy, administered for its relief, is signally efficacious. Children thus affected are usually the offspring of recently diseased parents, and exhibit other symptoms of constitutional lues.

The paralyses of the curiously selected ocular muscles, due generally to involvement of the third, sixth, and fourth pairs of nerves, coincidently or separately, have been explained by supposing that, before piercing the orbit, they traverse the base of the brain in intimate relation with its investing membranes, and the osseous surfaces which support it—a tract where neoplasms, gummata, and other syphilitic manifestations are of common occurrence. As causes also may be named, lesions of the nerve-centres in the gray matter of the cortex of the brain.

Secondary and rare primary lesions of syphilis affect the auricle, lobule, and post-auricular regions. Discharges occasioned by deeply situated, moist, and luxuriantly growing vegetations and mucous patches of the external auditory canal, are liable to be mistaken for those occurring in cases of simple otitis externa. Audition may be hindered by the growth of single and multiple osseous nodes, which compress or occlude the canal. Mucous patches, occasionally destructive by ulceration, occur also upon the lining membrane of the Eustachian tube, and the walls of the middle ear. The chief source of peril, however, to the latter, is the extension to this locality of grave nasal or pharyngeal disease, usually of the tertiary form (degenerating gummata and tubercles), whereby the whole or greater part of the organ of hearing is irreparably injured. When the tube alone is involved, there is hardness of hearing, a sensation of fulness in the ear, and perception of abnormal sounds. Occasionally the patient will seem to hear orchestral music, loud drumming, the sounds of machinery in motion, etc., for months without interruption. When the middle ear is involved, the pain is severe. The drum becomes uneven, less brilliant, injected and infiltrated. The sequelæ are, perforation, loss and loosening of ossicula, and osseous caries. Plastic forms of myringitis are accompanied by hyperplasia of bone and membrane, and isolated outgrowths in the form of polypi, nodes, and fibro-cellular neoplasms.

The pathology of suddenly occurring syphilitic deafness is unknown. Disorders of the labyrinth, cochlea, and of the malleo-incudal or incudo-stapedial joint have been suggested as efficient causes. The deafness of inherited syphilis is usually incurable and hopeless. In children born with normal power of audition, the result is, in general, deaf-mutism.

When syphilis occurs late in life, it is, as a rule, chiefly characterized by a lengthened period of incubation, and the development and progress of the initial and subsequent phenomena are relatively slow. The response to appropriate treatment in these periods is correspondingly tardy and unsatisfactory.

So-called "galloping syphilis," however, is encountered in the aged. Predisposing influences are intemperance, dissipation, anæmia, mental depression, "misery," improper hygiene, and concomitance of phthisis and scrofula. Phagedena is an early complication (though the gravity of an early symptom is no sure portent of future trouble), cutaneous lesions rapidly ulcerate, and phenomena usually expected in late or tertiary stages only, press close upon the date of infection. Thus bone disease, visceral complications, and retinitis with complete amaurosis or hemiplegia supervene in but a few weeks after the appearance of chancre; and the most intelligently directed and energetic treatment will scarcely avail to prevent disastrous results.

An important question concerns the admissibility of syphilitic patients to the married state. A favorable answer will rest upon the absence of actual symptoms, a certain period of immunity consecutive to the last manifestations, the benign character of the latter, the advanced age of the diathesis, and the subjection of the patient to a sufficiently prolonged specific treatment. Ignorant, feeble-minded, and vicious patients marry when syphilitic, even when affected with primary lesions; and the younger the syphilis the graver the danger for wife and offspring. The peril is great from a few months to three years after infection, but markedly diminishes from the fifth to the tenth year. In the matter of of time only, therefore, three to four years should elapse before marriage be permitted. But this period given, with it should be also considered the nature, duration, and results of the treatment pursued. As to the period of immunity from precedent symptoms, it should cover between eighteen months and two years. "Menacing" cases are those where there is frequent recrudescence of mild symptoms in benign syphilis, those where there is multiplicity, intensity, or gravity in the nature or tendency of symptoms, those which exhibit inordinate constitutional reaction in the presence of the infective process, those proving refractory to treatment, and those where the disease has a predilection for important organs or those essential to life.

Concerning the transmission of syphilis by inheritance, it is generally accepted that, when both parents are infected, what may be termed "intense" syphilis of the offspring will probably ensue. Nearly one-third of all such pregnancies result in abortion about the sixth month, or in still-births. It is also admitted that, when the mother alone is diseased, she will probably bear an infected child, and that the severity of symptoms exhibited in the product of conception will be proportioned to the length of the interval between infection and conception when no treatment has been adopted. When the father alone is infected, the child usually escapes the peril. Cases, however, are on record, observed by competent authorities, where an infected father and a healthy wife have produced an unmistakably syphilitic fœtus or child. In all cases it is conceded that, as the age of the diathesis in the progenitors advances, less and less diseased children are begotten, till those are born in whom no traces of lues can be distinguished. The question regarding the possibility of the transmission of syphilis through the utero-placental circulation may be considered as still *sub judice*. Unquestionably many women, infected with the disease after conception, abort with a dead or even macerated fœtus, the result merely of the depressing, rather than of the specific effect of the maternal disease. Such products of conception are not necessarily syphilitic. To be shown infected, they should differ from the aborted products of conception in healthy women. Again a mother, infected after conception, may bring into the world a child whose hereditary syphilis was derived from the father. But, according to the recently revived doctrine of *choc-en-retour*, the infected ovum or fœtus in the uterus of a healthy woman may impart to the latter the germs of its paternally derived disease. The entire question of utero-placental transmission requires for decision exhaustive knowledge of all the facts in the case of three individuals, father, mother, and child (the history of the pregnancies of a mistress or former wife of the husband is of additional value); and such knowledge is rarely had in reported exceptions to the rule. The recently reported cases, however, of *M. Zeissl* and *Hudson* seem to fulfil the indications for disproof.

It has been urged that the constitution of the mother who has borne a series of more or less infected children, and who yet herself betrays no evidence of the disease, is in some way modified so that she herself enjoys immunity from any subsequent danger. The modification is described as analogous to that wrought by vaccinia in contempt of variola. In support of this is urged the law of *Colles*, founded upon the well-nigh invariable immunity of the mother who suckles her syphilitic child, while the sound nurse, whose breast is given to the same child, is in imminent risk. We have ourselves pointed to the extreme probability that the healthy father of a child infected by a diseased mother would enjoy a like immunity; and in such case no modification of the paternal constitution could interfere. The facts ascertained point to a broad law of immunity for both parents as against the diseased child they have begotten.

Few instances of direct infection are on record, where the infant acquired the disease during the act of expulsion from the genital organs of the mother. The liquor amnii on the one hand, and the vernix caseosa on the other, furnish admirable safeguards against such an accident.

Hereditary symptoms of the living child usually appear about the third week, though often delayed till the fourth month. Cases are noted where the first symptoms are delayed during a longer period (twelfth to fourteenth month), but these as well as the very late manifestations of hereditary syphilis are justly regarded with suspicion. Syphilis of the third generation is of the rarest occurrence.

Cutaneous manifestations of the inherited disease are erythematous, papular, condylomatous (vegetating mucous patches), vesicular, pustular, furuncular, bullous, tubercular, and gummatous. The bullous syphilide, most precocious of these phenomena, often dates back to the sixth or seventh month of intra-uterine life, and is, as respects location, palmar, plantar, and digital; in hue from deep-violet to venous-red. Developing rapidly, the bullæ may increase to more than one centimetre in diameter, may coalesce, and then contain pus or blood, may have partially absorbed contents covered with brownish concretions, or may burst, leaving beneath a crateriform ulcer, this latter issue usually proving fatal. They rarely occur in successive crops, and when tardy in appearance co-exist with other lesions of variable type. Non-syphilitic pemphigus never appears suddenly upon the palms and soles; the lesions are rosy-red in tint, are larger, and contain an amber-colored serum which is either absorbed or results in a thin impetiginous crust whose fall leaves behind a delicate newly-formed epidermis. The disease rarely occurs before the fifteenth day of life, and may appear at any time during the course of the first year.

Mucous patches and gummata of mucous membranes are sufficiently common in hereditary syphilis. Among visceral lesions may be indicated splenic enlargements of two varieties, one dense, indurated, hepatic in color, the other friable and paler-hued, and, in the kidney, multiple, pin-head-sized, white, yellow, and red infiltrations, made up, as usual, of connective and round-cell tissue. These compress and destroy the tubules and lead eventually to colloid degeneration of the epithelium.

The testes in hereditary syphilis are less rarely invaded than the liver, but are often involved when the viscera are intact and yet unaffected and when there is extensive osseous and hepatic disease. The glands become voluminous, painless, and as hard as ivory, from a small nut to a pigeon's egg in size when very young children are affected. The scrotum is large, lax, and hyperæmic, with epididymis and cord usually unaffected. There is rare effusion into the tunica vaginalis. Microscopically embryonal bodies, like white corpuscles, are seen, which either build up variously-sized tumors traversing the trabeculæ, or are infiltrated throughout the connective tissue. Hypertrophy, sclerosis, or granulo-fatty degeneration may then obliterate the tubules, and complete atrophy or destruction of the organ result. The simpler forms of epididymitis, it should be remembered, are never followed by wasting of the testicle, the metastases of mumps only excepted. An hereditary affection of the ovaries from syphilis is also described.

Parrot, in France, and *R. W. Taylor*, in this country, have well described the bone-syphilis of infants. Atrophic changes occur upon the skull and the limbs, and are called gelatiniform, from their resemblance to fruit-jelly. The tint varies from garnet to rose, and deep citron to light maize, on the same bone. The medullary cells of the glistening and transparent marrow disappear, leaving only a vasculo-fibrillar meshwork. There is rapid decalcification of the hard parts, the spongy lamellæ melting away and becoming replaced by large vacuoles containing tissue like altered medulla. The chondro-calcareous form of atrophy affects the thin, so-called ossiform layer, between the osseous diaphysis and the cartilage. In syphilis, it becomes thick, irregular in its boundaries, and then denotes arrest of ossification. The portion of cartilage bordering the diaphysis, instead of being transformed into bone, remains simply eburnated and hardened. Vessels disappear, altered cartilaginous capsules with nucleated corpuscles replace the normal osteoblasts, and the diseased tissue extends to the spongy portion of the bone, from which it differs by a greater sponginess, friability, density, and a white color, like the chalk which it in other respects resembles. It may occur in isolated nodules in the centre of the spongy lamellæ, or, indeed, in the centre of a gelatiniform mass, for both forms of atrophy may co-exist in the same locality.

Both interfere with the solidity of the affected bone, producing a friability which may result in fracture at a distance of not more than from one to ten millimetres from the cartilage. When separation of the fragments is complete, the friction is liable to produce intra- and peri-osseous abscesses, and suppurative arthritis. "Syphilitic pseudo-paralysis" ensues, simulating that of nervous origin. There is sometimes complete inertia of the patient, with but little pain, the thoracic limbs hanging by the side of the trunk in a position of pronation, the pelvic limbs elongated, and oscillating with every movement. The muscles are intact, and there are swellings in the vicinity of the joints when there is abscess or notable displacement of a bony fragment. At times, gentle crepitation can be distinguished. The first variety of the second and rarer osteophytic form is the osteoid. The humerus and tibia, next the femur and ulna, lastly, the entire skeleton, are, in the order named, affected with the osteophytic development. The latter varies from one to four millimetres in thickness, most evidently on the posterior face and superficially seated. Occasionally the osteophyte is so intimately united to the diaphyseal surface that the line of demarcation is determined with difficulty. This line is formed of trabeculæ, which have a perpendicular, or very slightly oblique direction to the axis of the diaphysis. In cases, several layers can be distinguished, separated by medullary furrows or spaces, in which similar trabeculæ can be detected. The neighboring periosteum is thickened. The osteoid variety may exist at all ages, and is first encountered only before the sixth month. In it, interlaced and perpendicular trabeculæ are found, infiltrated with calcareous salts and surrounded by medulla. They are more rosy and yellowish, more easily broken, punctured, and cut than normal bone. Microscopically, they have not, like the latter, a systemic structure. They do not contain, like true bone, osteoblasts, regularly disposed in the fundamental substance around Haversian canals; there are triangular or polygonal corpuscles, recalling the stellate corpuscles of connective tissue, anastomosing by means of processes springing from them with the periosteum, whence they emanate, and with the analogous corpuscles existing in the medullary spaces. In the spongy, or rachitic form, which occurs very exceptionally before the sixth month, the osteophyte is formed of spongoid tissue, white, pearly, or slightly yellowish in color. It is moderately vascular, and contains but little marrow. There are a large number of intermediate varieties, from the simplest osteophyte to that where many layers, of variable structure and consistence, are so disposed that the centric deposits are hard from calcareous matter, and the peripheral layers are spongoid and sub-periosteal. The second form is, therefore, derived from the first, as intermediate conditions are demonstrable.

In the dactylitis syphilitica of the early months of hereditary syphilis, the lesions resemble those of the acquired disease, the deformity being produced by phalangeal enlargement, amenable to treatment, but producing, when unrestricted, lengthening, shortening, or thinning of the phalanx. The metacarpal and metatarsal bones are similarly involved.

The affections of the nervous system in hereditary syphilis are well described in the twelfth volume of this work.

In the treatment of primary syphilis, the best authorities are agreed in rejecting the destructive cauterization of the initial lesion. The simpler local dressings are to be preferred (black-wash, aromatic wine, calomel, iodoform in powder, etc.). Mercury, whose effect is speedy in hastening cicatrization of the ulcer and resorption of the sclerosis, is employed internally when indicated; but a mercurial course for the treatment of general syphilis is best deferred till the first explosion of the diathesis; exception being made for social emergencies, rebellious sores, and enormous chestnut-sized indurations. In the constitutional treatment of the disease, the hypodermic injection of mercury has lately attracted special attention. *Bamberger's* mercuric peptone, employed for this purpose, is prepared as follows: One gramme of meat peptone is dissolved in 50 cc. of distilled water. To this is added 20 cc. of a five-per-cent sublimate solution. The resulting cloudiness is removed by adding 15–16 cc. of a twenty-per-cent solution of sodium chloride. This preparation contains one per-cent of mercuric peptone; and has proved satisfactory in our hands in a limited number of cases. Other authors have similarly injected solutions of the bicyanide and biniodide of the metal. The merits of the best of these methods rest upon the introduction of a necessary quantity of the drug beneath the skin, in a condition in which it can be absorbed without further chemical change, and without the production of localized abscess at the site of the puncture. We have heretofore alluded to the fact that the hématimètre indicates an increase of the red blood-corpuscles in syphilis, when properly adjusted doses are administered, thus producing a tonic effect. In whatever way introduced, its presence in physiological secretions and excretions is demonstrable. The results of *Clevenger's* investigations have led to his belief in its mechanical effects. On the skin of a frog, to which five grains of blue mass had been administered twenty-four hours previously, numerous minute globules of the metal were seen under the microscope; blood-vessels were choked with it, aneurysmal and varicose pouches (supposed to be lymph-channels) were distended by it. Tubes, thus distended by globules increasing in size, collapsed on emptying their contents outwards. In one vein, a large globule of mercury lay motionless, while the blood-globules beat against it with as little effect as the water upon the stones in a brook, then changed position to pass it, and slid by it as rapidly as ever. Six metallic spheres measured the diameter of a single capillary, and could readily have traversed it abreast. The oleate of mercury, applied externally, furnished similar results. In another case, where the liver was choked with opaque sacculations, sections one-two-hundredths of an inch in thickness showed the unmistakable glint of mercury-globules. One gram of finely divided mercury in albumen was administered to a male frog. . In five hours globules appeared on its back, and dissection showed the intestines, renal, portal circulation, heart, and lymphatic channels beautifully injected with much more finely divided globules.

The so-called "tonic treatment of syphilis," employed by others before his popularization of it, has acquired favor by the efforts of *Keyes.* Any preparation of mercury may be used, but preference is given to the centigram granules of the protiodide. For a few days a minimum dose is administered, which is very gradually increased by the addition of one pellet, until mild irritant effects of the drug are induced (intestinal diarrhœa, lividity of the gums, fetor of the breath). One-half of this "full dose" is termed the "tonic dose," which may be continued with impunity for long periods of time. There is, however, good authority still for the *coup-sur-coup* method, by which strong impressions are made on the system and succeeded promptly by general tonic regimen and medication.

The toxic effects of the potassium iodide, whose extensive employment in syphilis requires no comment here, are coryza, œdema of the glottis, and erythematous, vesicular, papular, pustular, furuncular, bullous, and purpuric cutaneous lesions. We have collated about a score of cases where larger or smaller bullæ have appeared on the surface subsequent to its administration; and this fact should be remembered in the diagnosis of the rare forms of vesicular and bullous syphilides, when recourse has been had to the drug named. The presence of iodine has been demonstrated in some of the pus-containing lesions.

As a substitute for mercury, iodoform internally has failed to furnish satisfactory results. Tayuya (Dermophylla pendulina) having attracted attention in Italy, was employed by ourselves, and failed utterly in severe cases. The experience of others has borne the same fruit. Balneotherapy, when there was no mercury used, has proved of no greater worth. The relief obtained at the water cures and springs of Europe and America (in this country notably the Hot Springs of Arkansas) has usually been the result of vigorous medication at the hands of "physicians" resident at these resorts, a medication usually lasting only during the period of sojourn in these localities, and in that proportion liable to be spasmodic and correspondingly ineffective.

GLANDERS AND ANTHRAX.

BY

JAMES LAW, F.R.C.V.S.,

OF ITHACA, NEW YORK,

PROFESSOR OF VETERINARY SCIENCE IN CORNELL UNIVERSITY.

GLANDERS.

BIBLIOGRAPHY.

WILKINSON: Jour. of the Royal Agric. Soc., No. 50.—ROSSIGNOL: Traité d'Hygiène Militaire, Paris, 1857.—LARREY: Hygiène des Hôpitaux Milit., 1862, p. 63; Mittheilungen aus der Thierärztlichen Praxis, Berlin, 1876, 1877, 1878; Bul. de Soc. Cont. de Méd. Vét., January 13th, 1876; Nouveau Dict. de Méd. Vét., Tome VI., p. 470.—PERCIVALL: Hippopathology, Vol. III., page 197.—KRABBE: Deutsche Zeitschrift f. Thiermed. u. vergleich. Pathol., Bd. I., S. 286, 1875; Thierärztliche Mittheilungen der Münchener T. A. Schule, H. 17, S. 83; Repertorium der Thierheilkunde, 33 Jahrg., S. 139, 1872.—JOHN R. PAGE: Report on Glanders and Farcy in the Confederate Army.—JAMES LAW: Report on Glanders, New York State Agric. Soc. Trans., July, 1869.

ACCESSORY CAUSES.

The question whether glanders is ever developed *de novo* at the present day in the equine system is still undecided, and probably must remain so, because of the comparative ubiquity of the virus in all parts of the world, and the impossibility of excluding the idea of possible indirect contagion in any case which seems to have had a spontaneous origin. This much may be safely affirmed, that the comparative or absolute immunity of certain secluded countries for a number of years, the great prevalence of the disease in such countries as have an extensive traffic or movement in horses, and the definite suppression of the affection in places where well considered suppressive measures are rigidly enforced, show clearly that, if glanders is ever developed *de novo*, it is on rare occasions only.

As a working rule for the suppression of glanders, the doctrine of its development by contagion only, is by far the best, and just so far as the source of the malady has been sought in unhygienic conditions have suppressive measures failed to prove effectual. Thus at the beginning of the century, when Professor *Coleman*, of London, taught that most cases of glanders arose from lack of stable care, the losses from this affection in the English cavalry were very heavy. Later, when the mind of the army veterinarian was impressed above all with the doctrine that glanders was almost always the result of contagion, and that to avoid this every suspicious animal must be promptly destroyed, the total mortality was reduced to two per cent, and the discovery of a case of glanders became exceedingly rare and was considered as evidence of neglect (*Wilkinson*, Journal of Roy. Agr. Soc., No. 50). Up to 1836, the mortality among the French cavalry horses amounted to 180 to 197 per 1,000 per annum, and largely from glanders and farcy (*Rossignol*). More recently, when the doctrine of development *de novo* had largely given place to that of contagion, the mortality from glanders and farcy has fallen to two per cent per annum.

But with all this splendid show of results, there is a possibility and even a danger of erring on this side as well as on the other. The lamented *Gerlach* and his followers, in advocating *contagion* as the one efficient cause, seem to have largely ignored the accessory causes without which contagion acts only with a limited potency. Yet all acknowledge that, in addition to contagion, there must be a susceptibility on the part of the individual exposed. It may be added that this susceptibility of the individual may be enhanced or even acquired by unwholesome conditions of life, and such conditions, therefore, become no less operative in favoring the spread of the affection than is the simple fact of contact. It has been shown in Vol. III., p. 320, that of horses kept in ordinary conditions and inoculated with the virus of glanders, but twenty to thirty-five per cent contract the disease. But it is easy to find instances of animals kept in close ill-aired buildings, badly fed and overworked, in which the malady contracted by infection attacks all without exception.

Impure and Re-breathed Air as an Accessory Cause of Glanders.—As showing the evil results of re-breathed air, the statistics of the French army are most instructive. After 1836, a partial, but still very insufficient improvement in the ventilation of the military stable reduced the mortality from 190 to 68 per 1,000 per annum, of which fifty per cent was still from glanders and farcy (*Rossignol*, op. cit.). What is even more striking is that during the Italian war in 1859, 10,000 of these French cavalry horses were kept in barracks open to the external air, in place of closed stables, with scarcely any sickness and but one case of glanders (*Larrey*, Hygiène des Hôpitaux Milit., 1862, p. 63). In the British military movements, when the cavalry had to be transported by sea, in the confined holds of transports, there has almost always been a considerable manifestation of glanders and farcy, even in regiments that had previously been considered perfectly sound. The expeditions to Quiberon and to the Crimea may be quoted as striking examples. The recent expedition to Zululand appears to have been an exception and speaks strongly of the importance of the entire exclusion of the contagion, but not at all against the pernicious influence of impure air in the presence of the infecting material. As a concomitant of glanders in confined and badly ventilated buildings is the concentration of the infecting material in the atmosphere. The one infected animal gives off a certain amount of the virus, and according to the limitation of the space in which this can diffuse itself, will be the degree of saturation of the contained air with the morbid products and the efficacy of these on the animals condemned to breathe them. Further, as the number of cases increase, so will the amount of virus in the air and its infecting properties. As bearing on this subject, *Schmidt* says: When glanders appears in a large stud, the first necropsy shows old glanderous products with a limited number of more recent ones. This is repeated in several of the earlier cases, but as the victims multiply, the amount of recent deposits increase in the same ratio, and largely predominate over the chronic lesions (Mittheilungen aus der thierärztlichen Praxis, Berlin, 1878). While this seems to imply the infection of the stud by an animal having the disease in a chronic and occult form, it speaks even more strongly for the increasing virulence and severity of the malady as the number of cases increases and as the atmosphere of the stable becomes more thoroughly saturated with the poison. In the same publication it is recorded that over fifty per cent of the new outbreaks of glanders were clearly traced to the purchase of horses already suffering from this disease, while in almost every instance in which glanders has appeared on a farm without apparent cause, there has been one of the stud suffering for a length of time from some slight trouble with its breathing, or one that has remained in low condition in spite of the best of care, and that such animal has been found on post-mortem section to be suffering from pulmonary glanders. Here the one infected animal for a length of time fails to infect others, but as victims increase and the air becomes more charged with the morbid products the disease becomes more acute and virulent.

Cold, Damp Stables as an Accessory Cause of Glanders.—The following from *Leblanc* further illustrates the evil effects of unwholesome buildings: For fifteen years in the horses of the Company of Undertakers, Paris, he had scarcely seen a case of glanders, the last having been eight years before in the Alibert street depot, while in that at Fourneaux no case had occurred. In July, 1874, they were placed in a beautiful new stable where unfortunately the interior was dark, damp, and subject to constant currents of cold air. At the time of the change they were subjected to double work and were emaciated in consequence, and in three months 120 out of the 240 animals had contracted glanders (Bul. de Soc. Cent. de Méd. Vét., January 13th, 1876). These horses were constantly exposed to the possibility of infection while at work on the streets of Paris, but so they had been before the outbreak, and though this cannot prove a spontaneous outbreak, it proves most conclusively that the faulty stabling, overwork, and debility raised the affection to the dimensions of a most destructive scourge.

Debility from Low Feeding as an Accessory Cause of Glanders.—Among many instances may be quoted the following from *Bouley:* The horses of the administration of purified waters in Paris were fed liberally on oats and hay, and maintained fine health and condition. In 1849, the ration of oats was replaced by one of a new and much-lauded horsebread, insufficient for the demands of the animal system, and in less than a year 60 out of the 120 horses had contracted glanders and farcy, and were destroyed in consequence. The former ration was then restored, and as condition improved the number of new cases of glanders gradually diminished and the disease finally ceased (Nouveau Dictionnaire de Méd. Vét., Tom. VI., p. 470).

It would be easy to multiply instances in which debility from other causes—chronic and debilitating diseases, lack of sunshine, etc., etc.—

have laid the system open to attacks of glanders, and rendered that disease more than usually virulent. None of these can be shown to cause the development of the disease *de novo*, yet their importance in respect to prophylactic measures is none the less commanding. Where a malady like glanders tends to remain dormant for many months in an inappreciable form, as in pulmonary and other internal forms of the disease, its eradication from a district or country will be almost impossible so long as we allow those conditions which beget a special susceptibility, and insure the infection of every animal exposed to contagion. When, on the other hand, the conditions of life are such as obviate or remove this susceptibility, the extinction of this virus may be easily effected by the destruction of the diseased animals followed by a thorough disinfection.

QUESTION OF THE SPONTANEITY OF GLANDERS.

While it is impossible to demonstrate the development of glanders from insalubrious conditions of life, it is in the present state of our knowledge equally impossible to deny this. The almost universal prevalence of the disease is a strong argument in favor of its generation in a new locality without the introduction of the germs from without, yet with our past experience of contagion such evidence must be held as far from conclusive. It is but a few years since the medical world came to recognize the exclusively contagious character of small-pox and cholera out of the centre of Asia, or since veterinarians recognized that rinderpest, bovine lung plague, and aphthous fever are never spontaneous out of the centre of the eastern continent. The demonstration in the case of these has come from the long-continued and absolute immunity of certain countries that are not intimately connected with the infected ones, and by the observation that whenever any of these plagues has reached one of the countries in question, its advent could be clearly traced in the bodies of sick animals or some of their products, and that its progress through the newly invaded land has been slow and regular as new subjects have been successively submitted to contagion. Again there are many instances of countries and districts having kept clear of glanders for a length of time. In addition to those quoted in Vol. III., the following may be named: *Wilkinson* has shown that the occurrence of a case of glanders in the English cavalry, in the present day, is exceedingly rare and considered blameworthy (see above). *Charles Percivall* during an eight years' residence at Meerut and Cawnpore as veterinarian to the Eleventh Light Dragoons saw not a single case of farcy nor glanders. *William Percivall* in seventeen years' service in the First Life Guards had seen but one case and that from infection (Hippopathology, Vol. III., p. 197). *M. Saunier*, veterinarian to the King of Portugal, says that in his thirty years' residence no case had occurred in Lisbon. Later, during and after the peninsular war, glanders became a scourge of the country (Hippopathology, Vol. III., p. 197). *Krabbe* alleges that in the island of Bornholm with 7,000 horses, and in the Faroes and Iceland with 35,000, glanders is virtually unknown, the immunity being manifestly due to the very limited intercourse with the outside world and the rarity with which strange horses are introduced into these islands (*Deutsche Zeitschrift f. Thiermed. u. vergleich. Pathol.*, Bd. I., S. 286, 1875).

To the same purpose speaks the increasing prevalence of glanders and farcy wherever, owing to an active trade or to the movements of belligerent armies, the facilities for infection are increased. For Europe *Krabbe* (l. c.) states that, in the period from 1857 to 1873, in every 100,000 horses there became glandered yearly: In Norway 6, in Denmark 8.5, in Great Britain 14, in Sweden 57, in Würtemberg 77, in Prussia 78, in Saxony 95, in Belgium 138, in the French army 1,130, in the Algerian army 1,548. Setting aside the French armies, where the continued influence of the non-contagion doctrine must account for the heavy mortality, it will be seen that the losses are most severe in the central countries of Europe in which trade is most active and where the movement of animals and the opportunities for contagion are greatest. The evidence of the above statistics that the highest mortality occurred in Algeria is the more remarkable as it was formerly held that in this as in other dry semi-tropical countries glanders could not exist.

Of the influence of war an excellent instance is furnished by the war of the Rebellion. Exact statistics are wanting, but there is abundant testimony that the cavalry regiments of both sides were decimated by glanders, and the witness remains, for on the return of peace the sick horses were sold out and scattered everywhere and the infection still prevails from Maine to Montana and from the Lakes to the Gulf. In every large city scores or hundreds of glandered horses are kept at work and even in the country districts the disease is alarmingly prevalent (Glanders and Farcy in Horses, *John R. Page*, M.D., Charlottesville, Va.; Report on Glanders, *Journal of the N. Y. State Agric'l Soc.*, July, 1869, by *James Law*).

The Prussian official statistics show that during the Franco-Prussian war the losses from glanders were more than doubled. There were 959 cases for 1869–70, 966 for 1870–1, 1,729 for 1871–72, 1,721 for 1872–73, and 2,058 for 1873–74. *Hahn* says that in Bavaria the losses rose from 175 per annum to 390 per annum after the war (Thierärztlich. Mittheilungen der Münchener T. A. Schule, H. 17, S. 83).

DURATION OF GLANDERS IN THE OCCULT FORM.

The morbid products of this affection are occasionally confined to the lungs and other internal organs, so that their true glanderous nature fails to be recognized, and when horses are infected from animals suffering in this way, the cases are usually considered as of spontaneous origin. Any data showing the relative number and duration of such occult cases are therefore important. In 216 glandered horses dissected at the Veterinary College at Berlin, 10 had the lesions confined to the lungs. In 1875 a serious outbreak took place at Königsberg, starting from three horses that had for a length of time suffered from shortness of wind, but which on post-mortem section proved to be suffering from pulmonary glanders

(Mittheilungen aus der Thier. Prax. im Preuss. Staate, 1876). In Aix-la-Chapelle two horses showed the first outward symptoms of glanders ninety days after they had been in contact with a glandered horse (loc. cit., 1877). At Sigmaringen two horses showed glanders in January, having been exposed only on October 1st of the preceding year (loc. cit., 1876). In Berlin many cases are reported in which disease did not show itself for three months after exposure. In Potsdam glanders broke out seven months after the horses, the cause of the outbreak, had been bought. Finally in Posen two horses manifested glanders only 366 days after they had been exposed to infection (loc. cit., 1877).

In view of this frequent existence of glanders for a length of time in this occult and unrecognized form, and of the constant liability to infection through public drinking troughs, mangers, etc., etc., it is easy to conceive how a rigid investigation of every case of alleged spontaneous generation of glanders would only serve to show that it was the result of contagion mediate or immediate. But the question must still be left *sub judice.*

The frequent presence of these unsuspected glanderous products in internal organs demands a longer professional surveillance of an infected stud or stable than would otherwise be necessary. Not only should the survivors be considered suspicious for a year or more, but all animals dying on the place, from whatever cause, should be dissected to see whether or not they present in their internal organs the characteristic lesions of glanders.

PROGNOSIS AND TREATMENT.

That a number of horses recover from chronic glanders is unquestionable, while it must be allowed that a large proportion of the cases of alleged recovery are delusive, and after apparent convalescence, retain in some internal organs the morbid products, ready to burst forth anew whenever the subject is exposed to hard work, poor feeding, or unwholesome housing, yet it cannot be denied that a certain number recover permanently and afterward endure severe privations, and cohabit with sound horses for a long lifetime without manifesting or transmitting the disease. Acute glanders in all its forms is mortal. Chronic skin glanders (farcy) is well known to be amenable to treatment, and if the disease is confined to the skin the recovery may be permanent. The same may be said of certain cases of chronic glanders in which the lesions are confined to the nose and submaxillary lymphatic glands. Three such recoveries are mentioned in the Prussian statistics of 1876. I have now under observation a team which suffered from glanders seven years ago. The stable contained ten horses, one of which was found in the advanced stages of acute glanders with extensive nasal ulcers and deposits, enlarged and nodular submaxillary glands, and the characteristic sticky discharge. The two younger horses had the same nasal flux and glandular enlargement, but as yet no ulcers on the visible portion of the pituitary membrane. These two recovered under the internal use of arsenite of strychnia and bisulphites, and the local application of sulphurous acid and carbolic acid solutions to the nose, with iodine to the enlarged glands. They have since stood continuously with eight or ten sound horses and have neither shown glanders themselves nor transmitted it. While this much may be stated, there can be no question as to the impropriety of treating glandered horses when considered from the point of national sanitation. So many glandered horses have deep and unseen morbid products, as well as the superficial, and continue to diffuse the poison after an apparent recovery, that there should be no hesitation about the destruction of every glandered horse, ass, or mule, where this can be done officially for the national good.

INFECTION THROUGH EATING THE FLESH OF GLANDERED HORSES.

Lions, dogs, and other animals frequently contract glanders from the consumption of the carcasses of diseased horses. With men, however, such an occurrence is almost unknown, the immunity being probably due to the cooking of the flesh and the comparative insusceptibility of man to the contagion. Of the multitudes of men who habitually handle glandered horses, comparatively few fall victims to the disease. Of instances in which the flesh has been eaten with impunity the following may be noted in addition to that of *Decroix* (Vol. III.): *Staub* says the consumption of a glandered horse had no ill-effect (*Repertorium der Thierheilkunde*, 33 Jahrg., S. 139, 1872). *Ringheim* records that in the year 1808–9 one hundred glandered horses killed in the Danish military horse depots were accidentally used for the soldiers' rations, yet only one case of illness resulted. Here the immunity of the great mass of the army should go for nothing. The infection of the one shows that the intestinal canal of man, like that of animals, is receptive of the virus, and that under other circumstances the mortality might have been the rule and exemption the exception.

ANTHRAX.

BIBLIOGRAPHY.

Siedamgrotzky: Zur Kenntniss der Milzbrandbacterien. Deutsche Zeitschr. f. Thiermedicin u. vergleich. Pathol., Bd. I., S. 253, 1875.—**Klingelhöffer**: Zur Behandlung des Milzbrandes mit Carbolsäure. Berliner klin. Wochenschrift, 1874, No. 44.—**Bollinger, O.**: Ueber die Milzbrandseuche in den bayer. Alpen. Deutsches Archiv f. klin. Medicin, Bd. 14, S. 269, 1874.—**Bollinger**: Ueber die Bedeutung der Milzbrandbacterien. Deutsche Zeitsch. f. Thiermed. u. vergleich. Pathol., Bd. II., S. 341, 1876.—**Frankel** und **Orth**: 2 Fälle von Milzbrand

bei Menschen. Berliner klin. Wochenschrift, 1874, No. 22.—Hirschfilder, J. O.: Ein Fall von Pustula Maligna mit Mycosis Cerebri. Archiv der Heilkunde, 16 Jahrg., S. 376, 1875; Vergl. das erschöpfende Referat von Birch-Hirschfeld, v. über "Die neuren path. anat. Untersuchungen über Vorkommen u. Bedeutung niederer Pilzformen bei Infections-Krankheiten." Schmidt's Jahrb. der gesammten Medicin, Bd. 166, Heft 2, S. 169–223, 1875.—Koch: Untersuchungen über die Ætiologie der Wundinfectionskrankheiten, Leipzig, 1878.—Feser: Wochenschr. f. Thierheilk. u. Thiersucht, Nos. 24–5, 1879.—Cohn und Koch: Die Ætiologie der Milzbrandkrankheit. Beiträge der Biologie der Pflanzen. Zweiter Bd., zweites u. drittes Hft., 1876.—Davaine: Septicæmie et Anthrax. Rec. de Méd. Vét., March 15th, 1879.—Pasteur: Etiologie du Charbon. Rec. de Méd. Vét., March 15th, 1880.—Renard: Medication iodée appliquée au Charbon. Rec. de Méd. Vét., January 15th, 1879.—Pasteur et Toussaint: Le Charbon produit par des Aliments arrosés avec liquids de culture bacteridienne. Rec. de Méd. Vét., February 15th, 1879.—Toussaint: Charbon dans la Beauer. Avril 15th–30th, 1879.—Pasteur: Etiologie et prophylaxie de la Maladie Charboneuse. Rec. de Méd. Vét., Fevrier 28th, 1879.—Colin: Etiologie du Charbon, Janvier 15th–30th, and Fevrier 29th, 1879.—Toussaint: Du Mechanisme de la Morte Consécutive à l'Inoculation du charbon. Rec. de Méd. Vét., Janvier 30th, 1878.—Pasteur: Charbon des Poules. Rec. de Méd. Vét., Août 15th, 1878.—Pasteur, Chamberland et Joubert: La théorie des germes et ses applications à la médecine et à la chirurgie. Compt. Rendu de l'Acad. des Sciences, July 8th, 1877.—Arloing et Cornevin: Sur la Nature du Charbon symptomatique. Journal de Méd. Vét., Janvier, 1880.—Pasteur: Cholera des Poules, Fevrier 15th, 1880.—Pasteur: Le Charbon, Maladie de la bacteridie. Rec. de Méd. Vét., March 15th, 1878.—Megnin: Mouches Charboneuses, Novembre 15th, 1878.—Toussaint: Action phlogène du sang Charboneuse. Rec. de Méd. Vét., Mai 15th, 1878.—Toussaint: Théorie de l'Action des bacteridies. Rec. de Méd. Vét., June 15th, 1878.—Pasteur: Septicæmie et Charbon. Rec. de Méd. Vét., Mar. 15th, 1879.—Toussaint: Maladie Charboneuse causée par un nouveau vibrion aérobie. Rec. de Méd. Vét., July 15th, 1878.—Chauveau: Mouton de Algérie insusceptible au Charbon. Jour. de Méd. Vét., Oct. et Nov., 1879.—J. Cossar Ewart: Life History of Bacillus Anthracis. Quart. Jour. of Micr. Science, April, 1878.—Klein: Doctrine of Contagium Vivum. Q. Jour. of Micr. Sc., April, 1878; The Microphytes Found in Blood and their Relation to Disease.—T. R. Lewis: Q. Jour. of Micr. Sc., July, 1879.—Lewis: Flagellated Organisms in the Blood of Healthy Rats. Q. J. of M. Sc., January, 1879.—William Roberts: Doctrine of Contagium Vivum. Q. J. of Micr. Sc., Oct., 1877.—Wiltshire: Report on Red Water in Cattle in Natal. Veterinarian, August, 1877.—Greenfield: Lectures on Infective and Contagious Diseases. Vet., Jan'y and Feb'y, 1880.—Law: Malignant Anthrax in Livingston Co., N. Y. in 1875. Public Health Reports, Vol. II.—Law: Splenic Apoplexy. Jour. of the New York State Agric'l Soc., Nov., 1871.—Law: Malignant Anthrax. Nat'l Live Stock Jour., April, 1877.—Law: Diseased Animals as Human Food. Nat'l Live Stock Jour., Jan'y, 1879.—Mills: Anthrax Fever or Loodianate Disease. Veterinary Journal, April and May, 1880.—Grimshaw: Dublin Jour. of Med. Sc., August, 1878.

HISTORY.

The discovery of the Bacillus Anthracis, alleged to have been made by *Pollender* and *Brauel* in 1849, was only published by the former in 1855, while *Davaine* had already described it in 1850 (*Compt. Rend. de la Société de Biologie*). *Birch-Hirschfeld* says *Brand described* the microphyte in 1849, yet it was certainly *Davaine* that drew attention to it as probably pathogenic.

SUSCEPTIBILITY OF DIFFERENT GENERA TO ANTHRAX.

It has long been noticed that the herbivora are most susceptible to anthrax, the omnivora somewhat less so, and the carnivora least of all. In the closely allied bacteridian disease—septicæmia—*Davaine* found that three foxes fed for months on the bodies of infected guinea-pigs, maintained the most perfect health. The septic blood of the guinea-pig injected into the connective tissue produced an abscess in one fox, but left two unaffected. Similar blood thrown into the peritoneum of the fox proved fatal in twenty-four hours (Recueil de Méd. Vét., March 15th, 1879). Still more important is the observation of Prof. *Feser* that rats fed on a vegetable diet contracted anthrax readily by inoculation, while those confined to a flesh diet failed to develop it, though inoculated (*Wochenschrift f. Thierheilkunde und Thiersucht*, Nos. 24 and 25, 1879). The same rats that had successfully resisted anthrax while on a flesh diet, quickly fell victims to inoculation with anthrax fluids, after they had been restored to a vegetable diet. The relative immunity of the carnivora, therefore, is evidently not inherent to the genus as such, but is largely influenced by the nature of the food and the consequent modification of the blood and secretions.

It is noteworthy that though a pregnant animal may perish from anthrax, the bacillus has never been found in the blood of the fœtus nor in the placental liquids. *Bollinger* has proved the immunity of the fœtus by experiments on the sheep, and *Brauel* and *Davaine* on goats and rabbits (*v. Ziemssen's* Encyclopædia, 2d edition). It has been explained by considering the placenta as a "physiological filter," but is, perhaps, really dependent on the fact that the fœtus is practically a carnivorous animal, and hence, with its dependent membranes and fluids, insusceptible. Yet that the genus, independently of the aliment, has a potent influence, is abundantly proved.

Davaine inoculated rabbits and guinea-pigs with one one-thousandth of a drop of septicæmic blood, and invariably obtained a fatal result in the former, but no ill effect in the latter. Conversely he inoculated the same species of animals with one one-thousandth of a drop of anthrax blood, and lost all the guinea-pigs, but the rabbits recovered (Rec. de Méd. Vét., March 15th, 1879). Birds are much less subject to anthrax than are the domestic quadrupeds. *Colin* and others constantly failed to inoculate birds successfully, but *Pasteur* showed that they became at once susceptible to the poison when their temperature had been reduced several degrees by immersing the lower part of the body in cold water (Rec. de Méd. Vét., March 15th, 1880). The modification of nutrition and innervation as the result of exposure to cold is, probably, the cause of the increased receptivity to the poison, as a rise of temperature in the quadruped, in connection with rapid growth, plethora, insolation, etc., increases rather than diminishes the susceptibility to anthrax. Rabbits, too, with a normal temperature of 103 to 104° F., equivalent to that of many birds, are notoriously liable to contract anthrax in a fatal form.

It has been suggested that the immunity of fowls may be due to the largely carnivorous diet on which they subsist when running at large, but this fails to explain the predisposing action of cold.

As further bearing on this question of temperature, it may be stated that hot summers are those in which anthrax gains its greatest prevalence; yet no amount of cold is incompatible with its development. In a recent case in Livingston Co., N. Y., three horses and a cat died in midwinter, and while the temperature was below zero, after they had licked the frozen blood from a stone-boat on which the hide of an anthrax steer had been carried. Indeed, the extreme alternations of mid-day heat and midnight cold in spring and autumn are strongly predisposing, and it is likely that the disturbance of the balance of function and of the general health is the inimical influence. *Davaine* found that one one-thousandth part of the septicæmic blood necessary to kill a guinea-pig in winter will accomplish the same object in summer. He finds, moreover, that the septicæmic poison survives 100° C., but is rendered powerless by extreme cold. Anthrax poison is killed by a temperature of 42° C. (resting spores excepted), and is unaffected by a temperature of −40° C. −32°F. (Acad. des Sciences, Dec. 15th, 1879; Rec. de Méd. Vét., March 15th, 1879).

In connection with the varying susceptibility according to the species, it is claimed that the Algerian sheep in France have completely resisted the anthrax poison. *Chauveau* (*Jour. de Méd. Vét.*, Oct. and Nov., 1879) bought nine Barbary sheep, and inoculated them two, three, and even five times with anthrax blood, but failed to produce the disease in a single instance. He placed a sheep in cold water to reduce its temperature (after *Pasteur*), but it still successfully resisted the poison. *Chauveau* had previously observed that certain individuals among the native sheep enjoyed a similar immunity, and the question is pertinent whether such an immunity cannot be conferred by artificial means. In the light of recent observations of *Pasteur*, it seems not improbable that the insusceptibility may have been obtained through a previous subjection of the system to a modified and vicarious form of the same parasite. In another bacteridian disease (the so-called chicken cholera), he cultivated the bacillus in conditions that retarded its growth, and, by inoculating the product, produced a milder and non-fatal affection, which proved absolutely protective against any subsequent attack of the more fatal disease. Again, recent experiments suggest the probability that bacteria are destroyed by the products of the fermentations which they respectively engender (*Wernich, Bauman, Nencki*). This has its counterpart in the vegetable kingdom. Land that has been cleared of forest, grows trees of a different kind from that by which it was formerly occupied, and by which it is yet so abundantly seeded; fields laid down in clover soon become clover-sick; and all agriculturists realize the importance of a rotation of crops. If anthrax, as well as chicken cholera, is thus self-limiting, and if the morbid products laid up in the system by an attack will fortify it against all future exposures, then it will only be a question of mitigating the force of the poison, as *Pasteur* has claimed to have done in the case of chicken cholera, in order to furnish as efficient a prophylactic as vaccine is for small-pox.

OTHER CAUSES OF ANTHRAX.

That the hay made from grass grown on infected pastures and on the graves of anthrax victims is often a bearer of the poison is undoubted; but whether the poison circulates in the juices of such plants or is only deposited on its surface, like other dust, is as yet undetermined. In an outbreak on the yearly inundated banks of the Genesee River, near Avon, N. Y., in 1875, and on land formerly free from anthrax, forty steers died in a fortnight; and during the five years that have since passed, from four to ten cattle have died yearly, either on the pastures, or while being fed by the hay cut from them.

That hay containing the bacillus anthracis, or its germs, is most dangerous is shown by the death of sheep fed on such provender, after it had been sprinkled with water containing the bacillus (*Pasteur's* Report to the Minister of Agriculture, Sept. 17th, 1878; Rec. de Méd. Vét., Feb. 28th, 1879). *Pasteur* ascribes the results to the presence of wounds in the mouth or pharynx, through which inoculation could take place. *Cossar Ewart*, and *Burdon-Sanderson* reached a similar conclusion regarding mice, which ate anthrax products with impunity, if there were no sores about the lips or mouth (*Quart. Journal of Micros. Sc.*, April, 1878). Yet the frequency of intestinal anthrax, without any local manifestation of the disease in the mouth or throat, as would inevitably take place in case of inoculation, seems to show that the poison may reach the intestines in a potential condition in spite of the gastric secretions. Again the winter, the season of feeding on dry, hard, fibrous food, which would be likely to cause abrasions and wounds about the mouth, is the season that furnishes the fewest cases of anthrax.

Contagion through fodder implies the preservation of the poison in the soil, and according to present observations it may survive in the soil for an indefinite period. *Pasteur* has watered soil with anthrax fluids, and demonstrated the presence of the parasite a year later by microscopic examination, and by the fatal results of inoculation. In the outbreak at Avon, N. Y., above referred to, a victim was buried about ten feet from the bank of the Genesee River, and the first new sufferers, after a complete immunity of nine months, were six steers that had been observed to lick the fluids that oozed out on the river bank, between the surface stratum of sandy loam and the subsoil of clay. This happened in June, after a few days' heavy rain, and the disease subsided when the grave and vicinity were fenced in down to the water. *Feser*, *Koch*, and *Cohn* have drawn attention to the fact that in marshy localities there are to be found particles of a clear gelatinous substance, like the honey-dew of barley, and that these are composed of zooglœa masses of bacillus subtilis, which has the same microscopic characters and life history with bacillus anthracis. The conclusion seems inevitable that the latter survives and multiplies in the soil in the same manner.

NATURE OF THE ANTHRAX POISON.

That anthrax is a parasitic disease dependent on the presence of the bacillus anthracis is unquestionable, but that this parasite is the sole cause is by no means so well attested. It has been shown that in other bacteridian diseases, and notably in septicæmia, the addition of a second element is necessary to give pathogenic potency to the bacillus. Thus the bacterium termo, the cause of ordinary putrefaction, is so universally diffused that it is being constantly inhaled and swallowed, it covers exposed surfaces and falls upon abrasions and wounds, but it never induces septicæmia in a healthy subject, unless it is associated with the morbid product (sepsin, pyrogen) formed by its growth in a diseased system or in dead animal tissues. Even the combined product produced in decomposing animal matter, when inoculated on guinea-pigs, does not at first show the extreme virulence that it does after it has been inoculated from beast to beast through several generations of the poison (*Burdon-Sanderson*, lectures). Similarly in anthrax, *Paul Bert* (Compt. Rend. de la Soc. Biolo., p. 355, 1879) has been able to isolate two poisons from anthrax fluids, one of which, the bacillus anthracis, kills in thirty hours, and the second, unlike the bacillus, diffusible in liquid, kills in twelve hours. The bacillus died if subjected to compressed oxygen, while the associated poison survived. *Toussaint* had already (Rec. de Méd. Vét., May 15th, 1878) reached the conclusion that a distinct poison co-existed with the bacillus. Fresh anthrax blood inoculated on refractory subjects produced local inflammation and even abscess, whereas blood from sound animals caused no such phenomena. Filtered anthrax blood also determined a slight local inflammation, but without any constitutional symptoms. As no one has succeeded in separating this product of the bacillus from the bacillus itself, it cannot be determined whether the preliminary action of the former upon the tissues is essential to the virulence of the latter, or whether the virulent bacillus is not a sport determined by growth in decomposing animal products.

Since the publication of Vol. III. of *v. Ziemssen's* encyclopædia, the life history of the bacillus anthracis has been traced by *Koch*, *Cohn*, *Ewart*, and others. When cultivated on a warm stage, at 33° C., in albuminous fluids, like the fresh aqueous humor of a rabbit, the rods often became motile in the course of a few hours, and had alternate periods of motion and rest. In some instances a number of rods would cease moving and aggregate in a dense mass (zooglœa). Soon, however, the rods began to elongate, and in five hours had respectively increased to eighty or one hundred times their original length. These filaments will at times appear twisted up so as to form the most beautiful networks. If the temperature is reduced to 28° C., the growth of the filaments is delayed, and may not be completed so as to form spores under 36 or 40 hours. The filaments are at first perfectly hyaline, but soon the central protoplasm can be distinguished from the sheath, and quickly breaks up into segments about the length of the original rods, and these in turn break up into still smaller masses. Close to the lines of transverse division of the protoplasm, minute clear points appear, the first indication of the spores. As the spores increase in size and brightness, the filament breaks up into segments, each containing a single spore. The spores may escape in one of three different ways: 1st. The envelope may swell up, give way, and allow their escape. 2d. The segments formed by the segmentation of the filament may undergo granular degeneration and disappear, with the exception of the surviving spore. 3d. Without segmentation the filament may undergo granular degeneration and disappear, leaving the bright refrangent spores. The segments containing the clear refrangent spores may be mistaken for germinating spores, but are angular at the extremities, whereas the rods of germinating spores are rounded. The spores escape about the third day of cultivation as bright refrangent oval bodies with a gelatinous envelope and a diameter of $\frac{15}{25000}$ to $\frac{20}{25000}$ inch. On germinating, the spores grow at once into rods (bacillus), or according to *Ewart*, each first divides into four sporules from which the rods are developed. *Koch* describes the rods as formed from the gelatinous envelope of the spore, while the central protoplasm disappears. *Cohn*, *Klein*, and *Ewart*, on the other hand, say that the central protoplasm lengthens out into the rod, and that the gelatinous envelope appears to be used up in its growth.

If bacterium termo were introduced into the cultivation fluid before the formation of spores, the rods or filaments underwent granular degeneration and disappeared. Inoculation with liquids treated in this way did not produce anthrax. If the bacterium was introduced only after the formation of spores, it did not interfere with the life nor changes of the latter. If the anthrax liquid containing rods and filaments only were exposed to the air and underwent putrefaction, the anthrax poison was destroyed. If exposed to the air and putrefaction after the formation of spores, anthrax was still produced by its inoculation. Anthrax liquids kept at a moist temperature of 37° C. became non-infecting, but such liquids or tissues charged with rods or spores when dried at a heat of 38° to 40° C. remained infecting. Rods and spores remained active when sealed up in capillary tubes or when reduced to near the freezing point. These furnish important indications for the destruction of the bacillus. Free exposure to the air in the warm season secures the destruction of the rods or filaments in connection with the growth of the common septic bacterium. But the same conditions determine the germination of the spores into rods, unless they are actually dried up so that free exposure in damp, warm weather promises the destruction of the poison. While the poison will survive indefinitely in dry buildings, in the soil, and in other situations from which air is excluded, it cannot survive a continued free exposure with moisture and the decomposition of organic matter.

The spores are the most tenacious of life, yet the spores will germinate and pass into the more destructible rods, at a temperature (12° to 18° C.) at which the rods would remain unchanged.

Exaggerated estimates of the tenacious vitality of the resting spores appear to have been disproved by later observations. *Pasteur* found that they remained active after prolonged boiling and also after they had been subjected to a pressure of twelve atmospheres of oxygen. Dr. *Ewart* found that boiling for five minutes destroyed the poison. *Bert* and *Ewart* respectively found that the spores were inactive after they had been subjected for twenty minutes to twelve atmospheres of oxygen (*Quar. Jour. of Micr. Science*, April, 1878).

The cause of death in anthrax has been attributed to the action of the bacillus in robbing the blood of oxygen. *Pasteur* has shown that the parasite is aërobic; and the black, diffluent blood, the congested state of the veins, the right heart and lungs, the hæmorrhages and petechiæ in the anthrax carcase are indicative of death by suffocation. But to asphyxia must now be added the embolism of the capillaries in the organs affected by the bacillus. This has been especially demonstrated by *Toussaint* (Rec. de Méd. Vét., June 15th, 1878). The bacillus embolism in the tissues where the anthrax is localized is constant, but how much this may depend on the impaired or suspended nutrition consequent on the abstraction of oxygen from the blood may be matter of dispute.

That the blood of anthrax subjects is not only robbed of its oxygen, but is rendered incapable of absorbing the usual quantity has been shown by *Regnard* (C. R. Soc. Biol., 1879, pp. 317 and 465). A man weighing 80 kilograms (160 lbs.) accidentally inoculated took in but 7.924 litres of oxygen per hour, about one-third the usual quantity, and had his temperature reduced to 33° C. (91.5° F.). Similar results were obtained in a dog successfully inoculated with anthrax.

There is reason to suspect that under the name of anthrax several diseases due to distinct bacteria have been hitherto confounded. Swine-plague, chicken cholera, milk sickness, and others formerly confounded with anthrax are now known to be essentially distinct, though all associated with bacteria. *Arloing*, *Cornevin* and *Thomas* found in the diseased exudations and blood of symptomatic charbon (black quarter, bloody murrain) no rods, but corpuscles refractive at their centres and with dark outlines, and culture in a moist chamber with aqueous humor, and acid and alkaline human urine never led to the formation of bacillus anthracis, nor did thirty-four inoculations on horses, cattle, sheep, rabbits and mice lead to anthrax nor a single fatal result. This shows clearly that there is a form of black quarter that is not anthrax nor infecting, but the hastily-drawn conclusion that no such cases are inoculable is not therefore to be adopted. Anthrax infection by bites of insects is common in animals as in man, and in such cases the disease spreads from the point of inoculation in a diffuse manner closely analogous to black quarter. The following cases from my own experience are further corroborative: At Cockburnspath, Scotland, a yearling was attacked with black quarter and was bled, together with all the herd; the fleam used for the sick animal was not cleaned before it was used for the others, and next morning seven more of the herd had black quarter, the disease extending from the wound made in bleeding. At Brunt, Scotland, a shepherd skinned a calf that had died of black quarter and after washing and taking a turn among his sheep, castrated three litters of pigs, all of which died of anthrax. Black quarter is, therefore, to be considered as anthrax only when the bacillus anthracis is found in the fluids; in their absence it is usually an independent disease.

MORTALITY.

While *Bollinger's* estimate of seventy to eighty and upwards per cent is applicable to the worst types, it is by no means constant. At an outbreak in Livingston Co., N. Y., in a herd of two hundred head of cattle in which forty had succumbed in a fortnight, and fifty more were seriously ill at the time of my visit, and where three attendants had contracted malignant pustule from handling the victims, but two died out of the fifty actually sick, and no more were attacked.

PROPHYLAXIS.

In addition to the measures advised in Vol. III. of *v. Ziemssen's* Encyclopædia may be mentioned the movement of any herd in which anthrax has appeared on to a drier and better drained soil. In such soil the bacillus or spores come more freely in contact with oxygen and more speedily undergo destructive changes. There accordingly the poison is much less likely to be present or to persist if introduced. In the case at Avon, one hundred of the best steers were at once removed to a high dry porous soil and the rest restricted to the higher of the two meadows on which they had formerly grazed, and were shut out from all points at which the clay approached near to the surface. Then both herds were fed hay watered with a solution of carbolic acid and bichromate of potassa.

Burning of the anthrax carcasses and products is manifestly the most thorough measure, and this has been carried out by official order in the Bavarian Alps, the cremation of carcass, blood, fæces and straw being specially provided for; in cases where burning is impossible, these are to be buried not less than 9 decimetres (3 feet) deep. Those that cannot be at once buried are to be covered with earth to keep off the flies. This order further makes the removal of stock from the infected Alps obligatory under a penalty (*v. Ziemssen's* Cycl., vol. III., p. 527, German ed.). Since 1875 I have resorted to destruction by burning when it could be easily accomplished.

Infected fields, and, above all, graves should be shut up from stock and subjected to a rotation of cultivated crops, so that the frequent exposure of the soil may favor the destruction of the bacillus. If the soil or subsoil is impervious or if basin-shaped it should be thoroughly drained to secure a free permeation by air and the consequent sanitary results.

TREATMENT.

In addition to the measures advised in Vol. III. of *v. Ziemssen's* Encyclopædia, the following may often be resorted to with advantage: The deep cauterization of localized anthrax caused by accidental inoculation; the application over the sore and adjacent swelling of iodized phenol or other antiseptic; the internal administration of quinia, bichromate of potassa, and nitro-muriatic acid; and the hypodermic exhibition of quinia, iodide of potassium, and bisulphite of soda. In local anthrax and in the less acute cases in which the blood has not altogether lost its power of hæmatosis nor the tissues their normal function of assimilation, etc., recoveries may be expected. With much prostration, wine and other alcoholic and ethereal stimulants are valuable to tide over the period of depression.

PREVALENCE OF ANTHRAX IN MAN.

The transmission of this disease to man is far more frequent than is generally supposed. It is true we do not see such epidemics as that of St. Domingo in 1870, when 15,000 people perished in six weeks from eating the flesh of the dead cattle (*Placido Justin*), or those of the Russian steppes, where the prevalence of the disease has obtained for it the local name, Siberian Boil-Plague. Yet under the names of black erysipelas, carbuncle, diphtheria, etc., many cases occur that are easily traceable to infection from animals. About ten years ago Dr. *A. N. Bell* of the Sanitarian reported sixty cases. In the last decade I have been personally cognizant of thirteen cases, and four of these have occurred within the past two years in a town of 10,000 inhabitants and independently of factories working up hair or hides which prove the most frequent sources of infection. Independently of such factories the relative liability of animals and man may be estimated by the following: While 1,000 animals suffered and 650 died in the Bavarian Alps in 1874, but 7 men were attacked and all recovered. In an outbreak on the Genesee River in 1875, in which out of a herd of 200, 100 suffered and 40 died, 3 men were attacked with malignant pustule. Though the mortality in man is far inferior to that of animals, yet it is sufficient to demand the most rigid measures for the suppression of anthrax outbreaks, for the safe disposal of the carcasses and other products, for the seclusion, drainage, and cultivation of anthrax-infected land, and for the prompt treatment by iodized phenol or other caustic of any red spot with a dark centre on the skin of man. The disease should be made the subject of sanitary legislation, in the administration of which the veterinary physician must hold a prominent place.

NOTE ON ANTHRAX.—Since the above was sent to the printer, we have received the Journal of the Royal Agricultural Society for 1880, in which our anticipations with regard to the vicarious nature of mitigated anthrax poison are realized as the result of experiment. In 1878, *Burdon-Sanderson* and *William Duguid*, at the Brown Institution, inoculated guinea-pigs for several generations of the virus with the virulent products of anthrax cattle, and inoculated two yearling heifers and a calf with the blood or splenic pulp of the diseased rodents. All the guinea-pigs died, but all the ruminants recovered after three days' fever (temp. 103° to 106° F.), though the inoculated products contained myriads of bacilli, and proved fatal to all rodents experimented on. A second inoculation on the ruminants produced a similar but milder attack. This it will be observed anticipates *Pasteur's* experiments in the same direction with chicken cholera.

In 1879, *Greenfield* had charge of Brown Institution and carried on the above experiments with similar results in the main. With the blood or other virulent products of guinea-pigs that had died of anthrax he inoculated a steer, a six months' calf, and an old, emaciated, and pregnant cow. The first two recovered after a nine days' fever, but the cow died on the fourth day. Death was attributed to her poverty and pregnancy. The steer was inoculated four times in succession from anthrax guinea-pigs and a fifth time from an anthrax sheep, and the effects gradually decreased, the result of the fourth inoculation being practically *nil*, while that from the sheep a fortnight later developed a febrile reaction (temp. 104° F.). The liquids inoculated on these animals were constantly fatal to rodents.

Finally a sheep was inoculated with a cultivated anthrax fluid of the fourth generation, that had been passed through the guinea-pig, but with a fatal result. The experiments are being continued and further valuable results are to be expected.

HYDROPHOBIA.—TRICHINOSIS.

INTESTINAL PARASITES.

(CHIEFLY)

AMŒBA COLI.—ASCARIS MARITIMA.—RHABDITIS STERCORALIS.

BY

THOMAS E. SATTERTHWAITE, M.D.,

OF NEW YORK.

PRES. OF N. Y. PATHOLOGICAL SOCIETY; PATHOLOGIST TO ST. LUKE'S AND PRESBYTERIAN HOSPITALS.

RABIES AND HYDROPHOBIA.

BIBLIOGRAPHY.

**Fleming: Rabies and Hydrophobia. London, 1872.—Mittheil. aus der thier-
l. Praxis im Preuss. Staate, 21. Jahrgang, S. 100, 1874.—Kolesnikoff:
tralbl. f. d. med. Wiss., 1875, S. 853.—Bollinger: v. Ziemssen's Handbuch,
d III., 2. Aufl., 1876.—Moreau: Veterinary Jour., Dec., 1877.—Paul, J. and C.:
es on Hydrophobia (translated by Dr. B. Ellis). Ohio Med. and Surg. Jour.,
.—Feréol: A Case of Rabid Hydrophobia (translated by Dr. B. Ellis). Ohio
. and Surg. Jour., 1878.—Fahrer, Joseph: Brain, Vol. I., 1879.—Galtier:
ptes Rendus, 25 Août, 1879.—Hart: Prelim. Report on Rabies and Hydro-
ia. Brit. Med. Jour., Aug. 17th, 1878.—Friedreich: Deutsch. Arch. f. klin.
., XXIV., 1879.—Foote: Dublin Jour. of Med. Sci., Oct., 1879.—Bouley:
d'Hygiène Publique, 2, 1879.—Hammond: Dis. of the Nervous Syst., 1879.—
valewsky, P.: Memorabilien, XXV., 3, 1880.—Benedikt: Virchow's Archiv,
d LXIV., Hft. 4, S. 557.—Gowers: Trans. of the Path. Soc., London,
.—Curtis, T. B.: Bost. Med. and Surg. Jour., Nov. 7th, 1878.—Fitz and
ttuck: Bost. Med. and Surg. Jour., Aug. 29th, 1878. -Lindemann: Berl. klin.
hensch., 4, 1879.—Lancet, Oct. 18th, 1879.—Southam: Lancet, Sept. 6th, 1879.
mberg: Dis. of the Nerv. Syst., Vol. II.—Goddard: Med. Rec., Vol. XVI.,
, p. 152.—Trousseau: Clin. Med.—Lancet, Feb. 14th, 1880; Oct. 18th, 1879.
ed. Record, 1877, p. 472.—Med. Record, Aug. 9th, 1879,—Haggart: St. Louis
. Review, March, 15th, 1880.**

HYDROPHOBIA IN MAN.

INCREASE OF THE DISEASE.—According to *Bollinger*, the number of
ons in Bavaria that have suffered from the disease during the five
s from 1863 to 1867 has been on the average 13.8 per year, while
ing the seven years from 1868 to 1875 there have been 18 per year.
pper Bavaria alone, during the year 1875, no less than eight individ-
died of the disease.

PERIOD OF INCUBATION IN MAN.—This point has always been of
t interest and importance, but unfortunately varies between very
d limits. According to *Bollinger*, in six per cent it is between three
eighteen days, but is frequently from three to six months. In sixty
cent of all cases it is placed between eighteen and sixty-four days.
duration of the first or prodromic stage is usually not more than
nty-four hours, rarely two or three days or a longer period. Recently
a well-marked case, the patient, a mason, thirty-six years old (in
nner, of Schwabing, near Munich), killed himself on the second day
he attack. The period of incubation was two hundred and fifty days
llinger).

Prof. *Friedreich* gives the following instance which came under his immediate observation, where the period of latency was still longer. A boy, fourteen years old, was bitten in the hand by a terrier on July 14th, 1867. The dog, though not suspected of being rabid, was looked upon with distrust, and was placed under the care of a veterinary surgeon. He died a week later, and the post-mortem examination corroborated opinions formed by the after-history of the case. On May 4th, 1868, the boy, who had forgotten about the dog and the bite, began to show evidences of hydrophobia. He died on the 9th of the same month with characteristic symptoms. The duration of incubation was nearly ten months.

THE INFECTIVE QUALITIES OF THE HYDROPHOBIC POISON IN MAN.—*Paul Kowalewsky*, referring to an article by *Maurice Raynaud*, in which the latter states that the saliva of persons bitten by hydrophobic dogs can convey the poison to dogs, and probably also to other persons, and, therefore, urges the exercise of caution both among those who have to do with live hydrophobic patients and in making post-mortem examinations of hydrophobic subjects, instances the following facts which militate against the conclusions of *Raynaud:*

In June of 1876, a boy about nine years of age was brought to him. He exhibited characteristic symptoms of hydrophobia from a bite received three weeks previously. The phenomena manifested themselves on the third day and were very well-marked. While the patient was waiting in the ante-room, he attempted eating a pretzel, but was unable to, the fragments covered with saliva and partly masticated falling out of his mouth. An insane patient passing by seized the partly masticated food and swallowed it. The boy died on the following morning. The insane patient never exhibited any unfavorable symptoms.

A year later, another patient, a soldier, twenty-four years old, also in the third day of the attack, was brought under his care. This man insisted on kissing his physician, which he did at least ten times, and upon the mouth. No ill came of it. In the same year *Kowalewsky* injected the saliva of a hydrophobic dog into three other dogs. The result was negative.

On the other hand, a veterinary surgeon, M. *Moreau*, is said to have examined a dog that had died of rabies. He had an abrasion upon his finger. Three months subsequently hydrophobia developed and he died. Mr. *Southam* has been equally unfortunate with *Kowalewsky* in endeavoring to produce rabies in rabbits by the inoculation of hydrophobic saliva from the human being. According to Mr. *Sanders* many physicians have been known by him to have wounded themselves while making autopsies and yet no ill results followed, and yet it is said that in Vienna students are not permitted to be present in the dead house during the post-mortem examination of a hydrophobic case.

Shinkwin states that, according to the data at his command, there were no instances in which hydrophobia was communicated from man to man. It is not improbable, as *Trousseau* alleges, that there was an error in diagnosis and the disease engendered was traumatic tetanus or nervous hydrophobia.

LYSSOPHOBIA OR HYDROPHOBIA IMAGINARIA.—*Bollinger* gives the following instance, reported by *Weinlechner:* Soon after the death of a female operative who had died of rabies, a male friend (whose lip had at one time been bitten by her in sport and exhibited a scar), fell sick in the hospital with some hydrophobic symptoms. After a "sweat cure" he recovered from his lyssophobia, which it turned out to be.

In the *Lancet* there is another somewhat similar account: In August, 1877, when rabies was very prevalent, an excitable young man was seized suddenly with violent pains and other manifestations regarded by his medical attendant as indicative of hydrophobia. He took hot baths, and is now well. He had been bitten some two years previously by a spaniel, but had never remembered it until it was recalled to his mind by reading about hydrophobia in the newspapers. The dog was well at the time of the attack.

Fleming and *Trousseau* both recognize the imaginary or spurious form of the disease.

PATHOLOGICAL ANATOMY.

Dr. *W. R. Gowers* has devoted careful attention to this subject, and while he disclaims having noted any pathological changes that are distinctive of the disease, he has found a certain degree of uniformity in them. In the gray matter of the medulla and cord he observed great distention of the vessels. This condition was regarded as pathological, even after taking into consideration that death in hydrophobia is from asphyxia. The vascular distention was most apparent about the ganglionic bodies which lie beneath the floor of the fourth ventricle—a condition which is regarded by Dr. *A. W. Foote* as explaining the frequency of glycosuria in hydrophobia. In the regions of the nuclei of the glossopharyngeal, and hypoglossal and pneumogastric nerves, the perivascular sheaths were most markedly gorged with blood. The greatest intensity of pathological change coincided with the area that has been termed the respiratory centre. Dr. G., however, had seen similar collections in chorea in a dog. He lays weight, however, upon the site of these changes in hydrophobia.

Though peculiar changes have been described in the follicular glands, tonsils, and remaining parts of the throat, *Fahrer* asserts that in some well-marked cases these have been absent.

Bollinger, however, gives a case which came under his observation at the Munich Hospital, in October of 1875. There was moderate reddening of the pharynx, with ecchymoses in the vocal cords; the lungs also were full of blood, the right lower lobe being in a state of splenization, and there was croupous bronchitis. Both of these latter lesions, however, were caused by the entrance of foreign bodies into the lungs—in this case milk and chloral—which accidentally deviated from their proper course, while the patient was being artificially nourished.

Allbutt and *Coates*, according to *Fahrer*, describe changes in the nerve-cells and vessels, deposits of round corpuscles in the perivascular spaces, also granular degeneration, and even embolism, mostly about the respiratory centre.

Hammond has detailed the history of a case which he saw in June, 1874. The pathological appearances which were described in this case, and which concerned the nervous system, were briefly as follows :

In the cortical substance of the brain, an increase in size and number of the blood-vessels, with minute extravasations of blood. The first and second layers of cells had been replaced in large part by fatty matter. Between the two layers were amyloid corpuscles. The third layer was but little changed, and the others appeared not to have been involved. In the pons varolii, enlargement and thickening of vessels and extravasations were also seen. In sections made through the medulla so as to include the nuclei of the hypoglossals and pneumogastrics, there were also numerous extravasations.

Benedikt also has lately described peculiar inflammatory changes in the brains of rabid dogs, and in the case of a man. The morbid appearances are classed as granular degeneration, miliary abscesses and hemorrhages. *N. Kolesnikoff* has further described similar appearances, but neither of these alleged findings could be substantiated by Dr. *Augustus Forel*. His studies, conducted by excellent methods, and as yet unpublished, were made upon the brain of a hydrophobic man and several rabid animals (*Bollinger*).

On the other hand, Drs. *R. H. Fitz* and *Shattuck*, of Boston, in concluding their history of a case which had been under most excellent observers, and where the post-mortem and subsequently the microscopic examinations were conducted with unusual care, make the following statement : "In brief, then, the alterations were a diffuse cellular infiltration of the adventitia of the veins, venous injection and thrombosis, perivenous hemorrhages and miliary abscesses." The "hyaloid masses" which have been described are, they state, very often met with in healthy brains and cords hardened in chromic acid and its compounds, and are probably the result of decomposition independent of any pathological changes.

LYMPHANGITIS IN HYDROPHOBIA.—Dr. *Lindemann*, of Münster, describes a case of dog-bite seen by him first in December, 1875, which exhibits some new points of clinical interest.

The wound was inflicted in the latter part of August of the same year. The cautery was then thoroughly applied. On further investigation, the borders of the wound were found swollen, reddened, tender to the touch, and bluish-red streaks, corresponding to the course of the lymphatics, coursed upwards to the axilla. These phenomena disappeared the next morning.

Lindemann believes that, at one time early symptoms, and at another late ones, are explained in the following way: In the one case, the lymph stream carries the poison directly to the central nervous system. In the other, there is a local increase in the cellular elements leading to inflammation, absorption, and eventually the outbreak of the disease.

OFFICIAL PUBLICATION OF INFORMATION RELATING TO RABIES.—It is so plain that the public in general are very insufficiently informed as to the symptoms of hydrophobia in the dog, which indeed is a disease difficult of detection, that it seems scarcely worth while to mention the following case, of which there is a brief editorial notice in the *Lancet* (Oct. 18th, 1879), and yet the matter cannot be insisted upon with too much strenuousness. The owner of a dog, hearing him howl in the night, got up and administered a sound thrashing, getting in return a bite in the thumb. The dog, who had been "tumbling about" the day previously, was killed on the day following the bite, because of "its peculiar behavior." Hydrophobia set in and the man died. Rabid pets, it is said, have, in some instances, been nursed all through their illness and died in their owners' arms. The Government, it is very properly claimed, is largely responsible for death from this disease, which, according to our present notions, is preventable.

A general diffusion of information in reference to the symptoms of hydrophobia in the dog is urged upon the attention of the authorities.

Bollinger states that, to afford the public and especially dog-owners the possibility of self-protection, all cases of rabies should be made public in the most thorough manner. People, when once aroused, keep a better eye on the dogs and their condition of health, as every sensible dog-owner will be very much concerned in preventing his dog from getting bitten, and many will voluntarily see that their dogs are not exposed to contact with other dogs. Even if alarm is excited by the sensational publication of hydrophobic cases, the final result is good. All police regulations that are burdensome tend to diminish the number of dogs. Confinement of dogs, frequent inspection, muzzling and the like tend in this direction. In Nuremberg, in consequence of restraint and close inspection during the period from Dec., 1865, to July, 1866, the number of dogs decreased from 3,200 to 2,400 (a fall of about twenty-five per cent), but after the abolition of the restrictions the number increased again. In Munich, where from the year 1867 no general regulations as to the restraint or muzzling were imposed, the number rose from 4,200 in 1861, to 4,700 in 1868, and 7,300 in 1875 (*Bollinger*).

In Bavaria, according to recent calculations, there are 292,000 dogs to five million inhabitants.

As further reasons for the taxation of dogs, of which about eighty per cent are kept as matters of luxury, it is to be said that they are exclusively the intermediate bearers or breeding-places of various parasites, such as the tænia echinococcus and tænia cœnurus, which pass their larval stage in man and domestic animals, appearing again as the cœnurus cerebralis in the ruminants. The echinococcus and "staggers" disease are classed among the most frequent affections of the animals that are put to service in the country districts.

Muzzling can be dispensed with if the prescribed regulations against hydrophobia can be energetically carried out. At the close of 1863, when hydrophobia began to extend epidemically in Wurtemberg, the number of rabid dogs rose so that, in 1864, there were 171, but in the following year it fell to 27 in consequence of more strict observance of police regulations, and because dogs were not allowed to run about without muzzles. When these restrictions were removed, the number of rabid dogs again increased, so that, in 1861, there were 132 cases. In 1867 there were also 107 cases (*Bollinger*).

Every owner of a dog, as of any animal dangerous to the community, says *Bollinger*, should be, in all respects, answerable, legally, for the consequences of his rabidness, whether or not the sanitary regulations have been observed. When we have reached such a stand-point that the death of an individual from hydrophobia is regarded as a death resulting from negligence, and dog-owners are amenable to penal and civil justice for all the consequences of a bite, they will begin to realize that the keeping of a dog entails with it certain responsibilities.

The usual length of time, which varies between six weeks and three months, is too short, as in from one-fourth to one-third of the cases the period of incubation lasts more than six months. Suspected dogs should be quarantined for at least six months, or, which is better, killed at once (*Bollinger*). This latter measure has received the sanction of the municipal police in Paris, for under recent instructions from the minister of Agriculture and Commerce, an ordinance was issued by the Prefect of police on August 6th, 1878, directing that every dog or cat bitten, or suspected of having been bitten by a rabid animal, was to be peremptorily killed.

PROGNOSIS.

According to *Bollinger*, bites upon the face and head are most dangerous. Of fifteen persons bitten by a single rabid dog, in the instance he has given, eleven died of hydrophobia.

According to the report of the commission appointed by the *Medical Press and Circular*, in a total of 150 reported cases of hydrophobia, there were 103 deaths resulting from the disease; of the remainder 10 real recoveries were thought to be properly established, a very important finding certainly, as many authorities of the present day maintain that the disease is uniformly fatal. The 37 remaining cases were regarded either as examples of the spurious form or of some other affection, such as traumatic tetanus.

TREATMENT.

Since it is desirable to remove the virus as soon as possible, and cupping of the wound is generally regarded as a simple and effective way of accomplishing this object, it has been suggested that an ordinary wine-glass may be used for the purpose, a pen-knife making the scarifications.

Hydrate of chloral has been used subcutaneously by *Hanot* and *Cartaz* in Paris, but without success; the strength of the solution was one to five.

On one occasion, 13 grammes (nearly half an ounce) were injected, subsequently 20 grammes (5 drachms). The results were similar to those produced by the inhalation of chloroform.

In alluding to five cases, all fatal, that had been treated at the Manchester Royal Infirmary within a period of twelve months, the methods are given in the *Lancet* for 1879. They consisted of the hot-air bath (1), subcutaneous injection of chloral hydrate and morphia (1), chloral (1), curare and morphia (1), morphia alone (1). Chloral and morphia were thought to have very little influence on the disease, though masking the symptoms, but even for this object requiring almost poisonous doses. Curare is thought to be a very dangerous remedy, as in one instance respiration failed suddenly after two injections of one-quarter grain each at intervals of three hours. In the last instance, there was cyanotic discoloration and rapid, irregular and shallow breathing, which appeared shortly after one-sixth grain had been injected. The time-honored method of employing hot-air and vapor baths gives marked relief, and in the absence of success from drugs, is recommended for still further trial.

According to a report in a Russian journal, hydrophobia developed in a girl seven years of age, who had previously had diphtheria. She was much relieved in two hours and a half by inhaling three cubic feet of oxygen. This treatment was continued for ten days, in conjunction with the mono-bromate of camphor. Recovery ensued in two weeks.

The following remarkable case, which was published last year, is testimony to the efficacy of curare: July 28th, 1874, a servant girl was bitten in the heel by a spitz dog, presumed to have been rabid. Symptoms of the disease developed October 16th of the same year. Neither morphia nor inhalation of chloroform controlled the convulsions, and resort was had to curare by subcutaneous injection. At first 0.02 (gr. $\frac{1}{3}$) was administered and repeated in fifteen minutes. At 12 P.M. the intervals between the convulsions were two minutes, and the remedy was again tried. At 1 A.M., 2, 2.30 and 3.20 A.M., 0.03 ($\frac{6}{13}$ gr.) were administered. Marked symptoms of paralysis were now observed, and voluntary movements of fingers and toes executed with difficulty. At 3.30 there was some slight difficulty in respiration. This increased until 4.15, and artificial respiration had to be resorted to; at 4.23 the last regular convulsion occurred. At 7.30 she drank water without difficulty. At 8 A.M., the paralytic phenomena continuing, she was placed in a hot bath. The symptoms then abated, but at 9.30 P.M. it was thought advisable, in view of subsultus, thirst and photophobia, to renew the injection ($\frac{6}{13}$ gr.), which was done. The patient continued to improve from this time, and on December 3d she left the hospital, the wound of the foot being completely cicatrized.

Feréol concludes a paper of much research by enumerating some important facts which have not, he thinks, had due recognition. Among them are the following: The period of incubation of rabies lasts usually two months or less; occasional instances are on record where it has lasted eighteen months, and even, in one case, two and a half years. The symptoms are apt to vary with the case, but aside from the respiratory spasm, which is now pretty generally recognized as one of the most characteristic symptoms of the disease, there is a peculiar expectoration of bronchial froth, as he calls it. This material rises into the throat, and is then expelled by a spasmodic effort on the part of the pharynx. Then there is aërophobia, a kind of hyperæsthesia, where the skin is so susceptible to impressions that a breath of fresh air is sufficient to bring on convulsions.

TRICHINOSIS.

BIBLIOGRAPHY.

ZAUGGER: Wochenschrift f. Thierheilkunde u. Viehzucht, 1869, 55.—BOCKENDAHL: General-Bericht über d. öffentl. Gesundheitsw. d. Provinz Schleswig-Holstein für 1874. Kiel, 1875.-MITCHELL: Trichinosis in Otsego Township, Allegan. Co. Rp. Bd. of Health, Mich., 1878.—CRESSY, N.: Trichinosis. Tr. Vermont M. Soc., 1878; St. Albans, 1879, 33–39.—DEMARCHI, G.: La trichina spirale e la trichiniosi nell' uomo e negli animali. Gior. di med. vet., Torino, 1879, XXVI., 97-121, 1 pl.—ENGELHARDT: Beobachtung über nesterweises Vorkommen von Trichinen in einzelnen Muskeln des Schweins u. s.w. Cor.-Bl. d. allg. ärztl. Ver. v. Thuringen, Weimar, 1879, VIII., 126-129.—Inchiesta sulla stato della trichinosi in Italia. Gior. d. r. Accad. di med. di Torino, 1879, XLII., 248–250.—MAURI, F.: La trichinose en Espagne. Rev. méd. de Toulouse, 1879, XIII., 109-112.—Staatspolizeilichen (über die) Massregeln gegen die Trichinen-Krankheit. Bl. f. Gsndhtspflg., Zurich, 1879, VIII., 77-79.—ALVES BRANCO, JR., J. M.: A trichinose; fiscalisação do godo suino na capital; indifferença do governo e desleixo da camara municipal de Lisbon. Correir med. de Lisb., 1879, VIII., 89, 101.—BELLON: Trichines et trichinose. Alger. méd., 1879, VII., 146-153.—DEMARCHI, G.: La trichina spirale e la trichinosi nell' uomo e negli animali. Gior. di med. vet., Torino, 1879, XXVII., 645–671, 1 pl.—SILBERSCHLAG, C.: Die Trichinen-Krankheit und das Essen von rohem Fleisch. Deutsche Vrtljschr. f. öff. Gsndhtspflg., Brnschwg., 1879, XI., 232–234. -GABUZZI: Sur la trichinose à Constantinople. Gaz. méd. d'Orient, Constantinople, 1879-80, XXII., 27-30.—GILPIN, E. P.: Five Cases of Trichina Spiralis, etc. Am. Pract., Louisville, 1879, XX., 135-138.—DEMARCHI, G.: La trichina spirale e la trichinosi nell' uomo e negli animali. Gior. d. farm., etc., Torino, 1879, XXVIII., 97-145.—LABOULBÈNE: A propos d'une épidémie de trichinose, la prémière observée en France. Gaz. d. hôp., Par., 1879, LII., 163–178.—LOUGHRAN, E. H.: Trichinosis (3 cases). Am. M. Bi-weekly, Louisville, 1879, X , 169-173.—PETRI, A.: Tabelle über alle in Rostock im J. 1878 geschlachteten und auf Trichinen untersuchten Schweine. Arch. f. path. Anat., etc., Berl., 1879, LXXVI., 214.– Prophylaxe der Trichinose in Bayern. Aerztl. Int.-Bl., München, 1879, XXVI., 82–86.—GUZZONI, M.: La trichina e la trichinosi. Clin. vet., Milano, 1879, II., 77-101 —MADDREN, P.: Trichinosis. Proc. M. Soc. County Kings, Brooklyn, 1879, IV., 181-190.—Dictámen de la seccion de higiene publica y privada sobre "trichinas y trichinosis en España" An. r. Acad. de med., Madrid, 1879, I., 193–203.—Preussen Polizei-Verordung für den Regierungsbezirk Cassel; die mikroskopische Untersuchung des Schweinefleisches auf Trichinen betr. Med.-Gesetzgeb., Berl., 1879, V., 207.--ZIPPELIUS: Erfahrungen

bei der microscopischen Trichinenschau. Wochenschr. f. Thierh. u. Viehzucht, Augsburg, 1879, XXIII., 385.—WENDT, E. C.: Chronic Affections of the Muscles following Trichinosis. Med. Rec., N. Y., 1879, XVI., 319.—HÖBERLEIN: Der erste Fall von Trichinosis in Württemberg. Med. Cor.-Bl. d. Württemb. ärztl. Ver., Stuttg., 1879, XLIX., 201-215.—BOLLINGER: Zur Prophylaxie der Trichinose. Deutsche Ztschr. f. Thiermed., Leipz., 1879, V., 1-32.—EULENBURG, H.: Ueber die im Jahr. 1877 in Preussen auf Trichinen und Finnen untersuchten Schweine. Vrtljschr. f. gerichtl. Med., Berl., 1879, n. F., XXX., 175-181.—BADIA, S.: Conferencia pública sobre la triquina, etc. Encicl. méd.-farm., Barcel., 1879, III., 41-43.—POWER, W. H.: Report to the Local Government Board on an Outbreak of "Fever" that proved to be Trichinosis on board the Reformatory School-Ship "Cornwall." Lond., 1880, Eyre & Spottiswood, 15 pp., 3 pl., 9 diag. fol.—TUÑON, R.: La triquinosis en Sevilla. Gac. méd. de Sevilla, 1880, II., 73-93.—DICKINSON: British Med. Jour., May 29th, 1880.—KÖRNIG: Allg. med. Central-Ztg., Jan. 28th, 31st, 1880.

THE COURSE PURSUED BY THE PARASITE IN REACHING HIS FINAL RESTING-PLACE.—Most authorities maintain that the worms pass directly through the intestinal walls and the peritoneal cavity, and continue their migration until the muscles are reached. *Colberg*, however, has found trichinæ in the capillaries (*Deutsche Klinik*, 1864, No. 19) and *Heller* is inclined to believe that they use the vascular channels as a medium of conveyance. At any rate, he regards this mode of progression as the most probable one. The fact that the embryos of trichinous animals remain unaffected, has no weight as an argument against their being transported by the blood-streams. The embryos of rats with numberless filariæ in the blood are themselves free from filariæ. *Heller* observes that, in examining a rabbit artificially infected by feeding, he found a young trichina in the thoracic duct. The possibility that the parasite is conveyed through the lymphatics must therefore be entertained.

ŒDEMA AS A SYMPTOM OF THE DISEASE.—This late symptom is thought to be caused by the diminished forcing power of the heart, in conjunction with the diminished respiratory activity, caused chiefly by the presence of trichinæ in the diaphragm.

FREQUENCY OF THE DISEASE.—In Schleswig-Holstein, according to the report of *Bockendahl*, between the years 1865 and 1874 there were 68 hogs found to be trichinous, out of a total of 24,690, or 1 in 348. Of these, eighteen were imported from Denmark. Only a very small number are examined. In Altona alone, something like 300,000 hogs are annually killed, and most of them are exported. It is also said that in Schleswig-Holstein numerous American sides of bacon have been found trichinous. Of 5,673 that were examined, 47 were diseased; 1 in 121.

IS THE TRICHINA DERIVED FROM THE RAT?—Recent investigations having demonstrated that there is small ground for believing that the rats become infected by eating the dead bodies of one another, *Heller* has

NOTE.—It is obviously impossible, in a work of the present compass, to deal fairly with a topic such as trichinosis, of which it may truly be said the most valuable part has yet to be written. It is hoped, however, that the bibliographical references will in part make up for this deficiency. (S.)

had occasion to examine a great many dead rats in the vicinity of the Pathological Institute of Kiel, and he has frequently been able to substantiate *Gerlach's* statements, that they only eat one another when there is no other food. He has a firm belief that the disease originates with the hog and not with the rat.

TREATMENT.

With the view of destroying trichinæ in the stomach, *Heller* (*Deutsches Archiv f. klin. Med.*, 16, S. 626, 1875) gave salicylic acid dissolved in alcohol (144 grammes in 18 cc. alcohol, 4½ oz. in 4½ ʒ). Many experiments on rabbits with glycerin were unfavorable; he was not able to get into their stomachs more than 20 grammes (5 ʒ) of water and 40 cc. of alcohol (10 ʒ). Even with this quantity some of them died with severe spasms; the wall of the stomach was found subsequently to have undergone a sort of colloid softening.

RESPONSIBILITY FOR SELLING TRICHINOUS MEAT.—A decision of the Supreme Court in Germany (Nov. 3d, 1875) is quoted by *Heller*. A butcher who refuses to allow his hogs, which are slaughtered for purposes of food, to be examined microscopically, in case the use of the pork causes the death of a person, is amenable to punishment for a fatal negligence. The penalty can be enforced, even if the butcher did not know that the meat he sold was diseased, and even if there is no police regulation in force regulating the microscopic examination of pork. In instituting compulsory examination of meat, public slaughter-houses are said by *Heller* to be indispensable, as these institutions have long since been shown to be absolutely necessary in maintaining a strict supervision over the meat market (see *Gerlach:* Die Fleischkost des Menschen. Berlin, 1875, S. 137).

TRICHINOSIS ON BOARD THE CORNWALL.—During the month beginning October 23d, 1879, a number of boys on board the British schoolship Cornwall were attacked with symptoms similar to those of typhoid fever. Forty-three were reported on the sick-list. The symptoms were disturbances of digestion, marked diarrhœa, hemorrhages from the bowels, and petechial spots of a rose-red color on the abdomen. One patient died on the eighteenth day, and at the post-mortem examination trichinæ were found in the muscles. It was thought that the salt pork given to the boys on Mondays may have been trichinous, though the government officer who was intrusted with the examination failed to find any trichinæ in it.

According to a note from Dr. *Dickinson*, who is an authority in these matters, the disease was not trichinosis (*Brit. Med. Jour.*, April 3d, May 29th, 1880).

INTESTINAL PARASITES.

BIBLIOGRAPHY.

Tape-Worms.

Hixon: Slippery Elm Bark for Tape-worm. Ohio Med. Recorder, 1878, III., 256. —Masse, E., et Pourquier, P.: Le tænia inerme et ses migrations. Acad. d. Sc. de Montpel., 1877, V., 125–138, 1 pl.—Bonnet: Note sur le ténia inerme. Arch. de méd. nav., Paris, 1878, XXX., 398–401.—Kahanego, Z.: Budowa tasiemca nastroszonego (tænia perfoliata Göze) jake przyczyenk do anatomii i histologii ogniwców (cestodes) Pam. Akad. remiej w. Krakow, 1878, IV., 68–123, 2 tab.—Werner: Einige Erfahrungen über Bandwurmkuren. Med. Cor. Bl. d. Würtemb. ärztl. Ver., Stuttg., 1879, XLIX., 281-285.—Küchenmeister, F.: Quellenstudien über die Geschichte der Cestoden. Deutsches Arch. f. Gesch. d. Med. u. med. Georg., Leipz., 1879, II., 379-394.—Bérenger-Feraud: Note sur l'action tænifuge du sulphate de pelletièrine. Bull. gén. de thérap., etc., Paris, 1879, XCVI., 297-308.—Bérenger-Feraud: Pelletièrine pour l'expulsion du tænia inerme. Bull. gén. de thérap., etc., Paris, 1879, XCVIII., 8–19.—Le Vaillant: Emploi du sulphate de pelletièrine contre le tænia. J. de thérap., Paris, 1879, VI., 298.—Mégnin, P.: Nouvelles observations sur l'origine des ténias inermes; applications à l'hygiène (with discussion). Rev. d'hyg., Paris, 1879, I., 225, 331, 1 pl.—Küchenmeister, F.: Quellenstudien über die Geschichte der Cestoden. Deutsches Arch. f. Gesch. d. Med. u. med. Geog., Leipz., 1879, II., 308-317.—Malot: Observations de tænia inermis, dans des conditions très-rigoureuses d'étiologie et de traitement. Union méd. et scient. du nord-est, Reims, 1879, III., 1-3. —Brunighausen: Zur Bandwurm-Therapie. Berlin, 1879, XLVIII., 1177. Allg. med. Centr. Ztg.—Grynfeltt: Quelques mots sur le tænia, les tæniacides; et en particulier la pelletièrine. Gaz. hebd. d. sc. méd. de Montpel., 1879–80, I., 218–245.—Stein: Einiges neue über Bandwurmkuren. Wien. med. Presse, 1879, XX., 1439–1441.—Mégnin: Recherches sur l'origine des ténias inermes. Gaz. hebd. de méd., Paris, 1879, 25, XVI., 827.—Bartlett, J.: Observations of Forty Cases of Tape-worm. Chicago M. J. and Exam., 1879, XXXIX., 244–257.—Mégnin, P.: Nouvelles observations sur le developpement et les metamorphoses des tænias. Compt. rend. Acad. d. sc., Paris, 1879, LXXXVIII., 88.—Elben, R.: Ein gutes Geheimmittel gegen Bandwurm. Med. Cor. Bl. d. Würtmb. ärztl. Ver., Stuttg., 1879, XLIX., 225.—Féréol: Ladrerie généralisée chez un homme ayant rendu un tænia; complication de diabète sucré. Courrier méd., Paris, 1879, XXIX., 257.—Besnier: Observations sur divers points, etc., de l'histoire du bothrocéphale et observations d'un cas, etc. Union méd., Paris, 1879, 3 s., XXVIII., 285-291.—Mégnin: Sur l'origine des ténias inermes. J. d. conn. méd. prat., Paris, 1879, 3 s. i., 472. Also: Neue Beobachtungen über die Entwickelung u. die Metamorphosen der Tænia, etc. (Translation). Rev. f. Thierh., Wien, 1879, II., 129-131. —Masse, E.: De l'origine du tænia inerme de l'homme. Bull. gén. de thérap., etc., Paris, 1880, XCVIII., 145–157.—Thomas, H. J.: Pumpkin-seed for Tapeworm. Chicago M. J. and Exam., 1880, XL., 173–175.—Pratt, G. B.: On Tapeworms. Chicago M. J. and Exam., 1880, XL., 77–81.

Lumbricoids.

Hutchinson, J. A.: Supposed Congestive Chills Caused by Lumbrici. Med.

and Surg. Report., Phila., 1879, XLI., 393.—Dorvan: Epilepsie, vertiges, accidents gastro-intestineux dus à la présence de vers ascarides lombricoides chez l'adulte. Arch. de méd. nav., Paris, 1879, XXXI., 72–74.—Cassali, T.: Un caso di elmintiasi con fuoriuscita di ascaridi lombricoidi dall' ombellico. Raccoglitore med. Forli, 1879, 4 s., XII., 281–291.—Sangalli: Perforation de l'intestin grêle intact par des ascarides durant la vie du malade. Compt. rendu, Genève, 1878, 247–250.

Anchilostomes and the Anguillule.

Bozzolo, C.: L'anchilostoma duodenale in Torino. Osservatore, Torino, 1879, XV., 369.—Grassi: L'anguillula intestinalis. Gaz. med. Ital. lomb., 7 s., V., 451–454.—Grassi, B.: Intorno ad una nuova malattia del gatto, etc. (anemia da anchilostomi). Gazz. med. Ital. lomb., Milano, 7 s., V., 451–454.—Normand: Comptes rendus, LXXXIII., No. 5, 1876, p. 316.—Bavay: Ibid., No. 15, p. 694.—Davaine: S. 966, II. Aufl., 1878.—Ciniselli, G.: Contributo al indagini sugli anchilostomi. Ann. univ. di med. e chir., Milano, 1878, CCXLV., 389–391.—Lonsino: Sull' anchilostoma duodenale. Imparziale, Firenze, 1878, XVIII., 611–618.—Grassi, B.: Sovra l'anguillula intestinale. Arch. per le sc. med., Torino, 1879, III., No. 10, 1–14, 1 pl.

For information on matters relating to intestinal worms in general the reader is referred to the following articles:

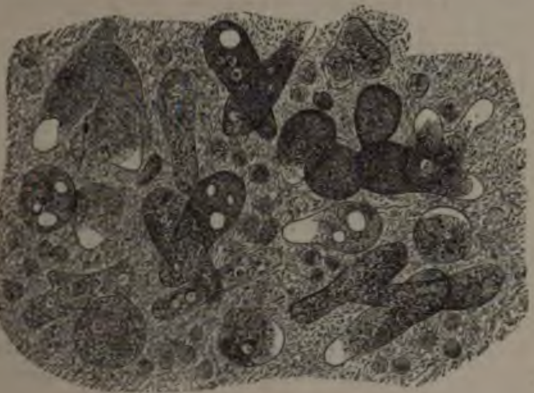
Lloyd: Knot of Intestinal Worms. Trans. Path. Soc., Phila., 1878, VII., 24. —Wiart: Vers intestinaux. Année méd., Caen, 1878–79, IV., 13, 29, 44, 56.—Scheuthauer, G.: Käsig zerfallende Herde in der Leber eines Knaben, bewirkt durch Spulwümer der Lebergallengänge. Jahrb. f. Kinderh., Leipzig, 1878, n. F., XIII., 63–69.—Goabert: Des vers chez les enfants. Paris, 1878, Dion, 163 pp.—Grassi, B.: Contribuzione allo studio dell' elmintologia. Gazz. med. Ital. lomb., Milano, 1879, 8 s., I., 115–154.—Lebert, H.: Entozoen. Handb. d. Kinderkr. (Gerhardt), Tübing., 1879, IV., 2 Abth., 277–336.—Cil: Entozoarios; un ejemplar de ellos sin nombre (caso práctico). Sentido catól., Barcel., 1879, 1–3.—Ransom, W. H.: Intestinal Worms in Dis. of the Intest. and Periton. 8vo, New York, 1879, 153–175.—Grassi, B.: Enterocolite con parassiti nota preventiva. Gazz. med. Ital. lomb., Milano, 1879, 8 s., I., 286.—Joseph, G.: Ueber die in den krainer Tropfsteingrotten einheimischen freilebenden Rundwürmer (Nematoden). Jahresb. d. schles. Gesellsch. f. vaterl. Cultur, 1878, Bresl., 1879, LVI., 76–78.—D'Auriol: Des vers intestinaux chez les enfants. Rev. méd. de Toulouse, 1879, XIII., 17–27.—Herrick, S. S.: A Case of Periodic Worm Fever. N. Orl. M. and S. J., 1879–80, N. S., VII., 850–852.—Cahis y Balmañya, M.: Catalépsia verminosa. Bol. med. farm. d. Litoral, Barcel., 1879–80, I., 26–34.—Lambl: Aus dem Franz-Josef Kinderspitale, I., S. 362.—Lösch: Virchow's Archiv, 65, S. 196, 1875.—Malmsten: Henschen Upsala läkerofor., etc., X., S., 120, 1874.—Nord. med. Ark., VII., No. IV., S. 17.—Treillé: Arch. de méd. nav., 24, p. 129, 1875. —Jhrb., 1875, I., S. 424.—Leuckart: Parasiten, II., S. 877–1816.—Lenoir: De la diarrhée chronique de Cochinchine. Thèse, Paris, 1864.—Quetaud: Arch. de méd. nav., 23, p. 197, 1874.—Antonie: Thèse de Paris, 1873. (Jahresb., 1875, I., S. 423.)

Of the fifty or more animal parasites that are found in man, twenty-four, according to very recent studies, have their habitat in the digestive tract. Of these three have been discovered within a few years. They are the *amœba coli* which is classed among the infusoria, the *ascaris maritima* and the *rhabditis stercoralis;* both latter are round worms. The last is of especial interest as, according to *Normand,* it causes the endemic diarrhœa of Cochin China.

Regarding the better known intestinal parasites, but little advance has been made in recent years.

THE AMŒBA COLI.

Heller states (1876) that there has been only a single article on the amœba, and this an unsatisfactory one by *Lambl.* It is said that in this instance amœbæ were found in a child two years old that died of enteritis. *Lösch,* however, has published a case of chronic dysentery, in which enormous quantities of amœbæ were found. These animacules were of a rounded, oval, or pear-shaped form; their breadth varied between 0.02 and 0.035 mm., while their length reached 0.06 mm. The body consisted partly of coarsely granular, partly of hyaline protoplasm, and contained transparent, rounded nuclei and numerous hyaline vesicles (Bläschen). The protoplasm also occasionally inclosed white and red blood-corpuscles, granules of broken-down intestinal epithelium, bacteria, etc. After enemata of cinnabar, the protoplasm was also seen to contain particles of cinnabar. The number of the hyaline vesicles varied between one and eight and even more; usually they were as large as the nuclei, but sometimes much larger, and even half the size of the animal. The amœbæ were almost continually in motion, executing changes of form and place; these latter were so rapid and peculiar that there was no possibility of confounding them with other normal or diseased cellular bodies undergoing amœboid movements.



Magnified 500 times (LŒSCH).

Lösch describes these movements in the following terms: "Take any spot that you choose upon the surface of the body, and you will see a flattened, rounded, bright, transparent prominence, limiting itself sharply from the surrounding granular protoplasm. Now it is either withdrawn or rapidly increases in size and spreads itself out until at length a fungus-like projection is formed (pseudopod), whose length sometimes equals the diameter of the rest of the body. This process may now be withdrawn, to appear again at another spot, or the granular protoplasm may suddenly pour itself into the process, and fill it more and more. In this way the form of the whole animal changes, becoming a long or perhaps irregular, oval, if several processes are thrown out at the same time. These projections are always blunted, never pointed or filiform. In comparison with the amœboid changes in the white blood-corpuscles, the former are very rapid. In a single minute, four or five may be withdrawn and again thrown out. The animalcules, in executing these movements, usually remain a considerable time in one place, then putting out a pretty large transparent process, gradually crawl a little further on. Meanwhile they permit the granular protoplasm to rush into it, and then draw after them the hinder part of their body. The absolute changes in place occur with comparative slowness, as it takes fully a minute for them to progress the length of their own body. The amœbæ were found in great quantity in the yellowish-white and grayish red masses of mucus and pus which were very abundant in the fæces, and yielded a considerable sediment on standing." The patient, a peasant 24 years of age, had a long-continued and very exhausting diarrhœa in 1871, but recovered. In 1873 he was taken sick again with the symptoms of a severe dysentery. Notwithstanding various methods of treatment, the disease progressed with some slight show of improvement. As there seemed to be some slight relation between the number of the amœbæ and the severity of the disease, an attempt was made to destroy the animalcules with quinine. Experimentally a solution of quinine sulphate (1–5000) was found sufficient to effect this object in less than a minute. The patient was then given, morning and evening, an enema containing sulphate of quinine 3.5 gm. (ʒ i) in water one pound, at the same time quinine was given internally to the amount of six grains. Under this treatment the amœbæ rapidly disappeared, so that after ten days it was impossible to find them, and meanwhile the condition of the patient had improved notably. A few days later, however, when the quinine treatment had been suspended, the amœbæ again appeared, and with them there was a return of the severe symptoms, which gradually led to very extreme exhaustion, notwithstanding various other methods of treatment; seven weeks later, an attack of pleurisy supervened, which proved fatal, in conjunction with cheesy pneumonia and the formation of cavities. Contemporaneously with the appearance of pleurisy, the intestinal discharges underwent remarkable changes: the amœbæ disappeared at once, owing probably to the abnormal metamorphoses in the intestinal contents consequent upon the fever. The post-mortem examination showed fatty heart, caseous infiltration with cavities in the apices of the lungs, and fibrous pleuritis. The mucous membrane of the ileum in its lower third was markedly hyperæmic and exhibited a coating of clayish consistence, with some flat irregular ulcers. The large intestine in its upper part was also decidedly hyperæmic, œdematous, and covered with diphtheritic deposits; it was also in part broken down; lower down the mucous membrane was much thickened, swollen, reddened, though only in part, and marked by numerous cicatrices and cicatrizing ulcers.

In order to reach some conclusions as to the relations between the amœbæ and the observed morbid phenomena, *Lösch* had three dogs injected, per os et anum, with from 30–60 grammes (1–2 ounces) of recent discharges containing amœbæ. He repeated the injections for three successive days. Prior to this experiment, he had injected a fourth dog with enemata containing croton oil and thus caused an intense enteritis. The object he had in view was to determine whether the amœbæ had the property of keeping up an existing inflammation. This experiment, as also two of the former, proved negative. In the single case where the result was positive, the injections had, for their first result, vomiting, diarrhœa, loss of appetite, and malaise; these phenomena disappeared in two days, and the dog seemed to recover. On the eighth day, however, t was observed that there was a little ball of bloody mucus, the size of a pea, in the otherwise normal fæcal masses. This little ball had proved to be permeated by a great number of living amœbæ; the quantity of mucus now increased rapidly in amount, and some was found adhering in several places to the fæcal balls. As in the course of two weeks no further changes were present, and the quantity of the mucus did not increase, the animal was killed (eighteen days after the last injection). At the post-mortem examination, the mucous membrane of the rectum was found reddened in places, irregularly swollen, and covered with tough, bloody mucus. In three places, there were slight ulcers, rounded in form, 4–7 mm. in diameter, and surrounded by swollen and hyperæmic mucous membrane. The bases were uneven and of a dark-red color. The submucous tissue was also red, swollen, infiltrated, and dull in color. The mucus of the rectum, together with the blood of the ulcers, was thickly permeated with amœbæ; otherwise there was nothing abnormal.

Lösch thinks it probable that the patient was first taken sick with dysentery, and that the morbid state was maintained by the additional presence of amœbæ; he had no information as to the source of the amœbæ. In similar cases, it is recommended to practise continuous treatment with quinine, and particularly to wash out the intestine after the manner of *Hegar*.

BALANTIDIUM COLI.

This little animalcule, which has been called paramœcium coli by *Malmsten*, has now been observed ten times in Norway. In all, it has been described sixteen times thus far: in females, seven times; in men, eight, and once in a person whose sex has not been given. Quite recently it has been observed by *Treillé* in a slight epidemic of tropical dysentery on the Volta, between China and Cochin China. In nine of the fifteen cases, the intestinal discharges were examined, and in six the little animals were found. *Davaine* confirmed the results. Information is very meagre regarding it.

THE ASCARIS MARITIMA.

This is a new variety of ascaris, of which the description of *Leuckart* is borrowed, as his is the only example that as yet has come to hand. The fructified female is forty-three millimetres long and one millimetre broad. The extremity of the tail, which measures half a millimetre, is shaped like a long and rather slender ten-pin. The suction apparatus is small (0.16 millimetre broad and 0.065 millimetre high); the head one millimetre behind is half a millimetre broad.

The cuticula is somewhat tumefied on either side behind the suction apparatus. The lips have deeply raised posterior surfaces, so that the two folds, together with their teeth, are sharply separated from one another. Of its history and habits but little is definitely known.

RHABDITIS STERCORALIS.

In the so-called Cochin China diarrhœa, a destructive endemic disease in Cochin China, *Normand* found a round worm, which he terms for the present "anguillule stercorale." *Bavay*, on the other hand, has named it "rhabditis stercoralis." According to present information, the parasite infests the patient, both in its sexually complete and in its larval state. As the former, it is found in the stomach, in the entire intestine, in the pancreatic duct, in the ductus choledochus, in the biliary passages, and the gall-bladder. The female, one millim. long and $\frac{4}{100}$ millim. broad, is cylindrical in form, though tapering slightly anteriorly and sharply posteriorly. The head is furnished with three lips imperfectly separated from one another. Of these, a single one is tri-lobate. The œsophagus occupies a fifth of the entire length of the body; anteriorly it is long and straight, while the middle portion, smaller in size, separates it sharply from the posterior ovoid pharynx; in the latter is a valvular apparatus. The gut is somewhat dilated anteriorly; it opens laterally near the root of the tail; its walls are not easily seen; on either side it is bordered by a light yellowish-brown gland. In the female, these parts are more or less displaced by the accumulated ova.

The vulva is situated a little behind the middle of the body; it leads into a uterus which extends both forward and backward, which, in the fructified condition, contains from thirty to fifty ova. The latter, at first brown, and then yellowish, disclose the embryo; sometimes the embyro even slips out into the uterus.

The male is far smaller than the female (about one-fifth the size); the testicle surrounds the intestinal and glandular appendages, and opens near the anus; here there are two small curved spicula, slightly thickened at their base. Behind them is a very delicate umbilicated process, which is shorter, but broader than the spicula. The tail is shorter than in the female, and turned to the same side as the spicula. The males are more rarely met with than the females.

In the embryos, the digestive organs are scarcely visible, the intestine is very short in comparison to the œsophagus, the uterus is not to be seen. These animals are most frequently met with in the undeveloped condition. They are then one-third millim. long and one-fiftieth millim. broad; the intestine contains oil droplets from the milk which the patients have taken; the uterus is only visible as a vesicle; the vulva is not as yet open. In five days, the worm is said to have attained its full sexual maturity, if the conditions have been favorable.

According to the first communication, to which the second has no relation, the worm appears,in its larval state, to develop in the intestinal wall, or in the follicles of the intestine. *Normand* saw one inclosed in an envelope; its substance appeared to be formed of nucleated corpuscles aggregated together in the form of irregular cylinders.

The worm has thus far been found in at least thirty cases of Cochin China diarrhœa.

(The above two communications are far from being satisfactory; a more complete account with drawings is expected.)

THE COCHIN-CHINA DIARRHŒA.

This endemic disease, one of the most fatal of Cochin China, killing the majority of those attacked, is said to be caused by the worm just described. It has been most thoroughly studied by French military surgeons, and is described as having a chronic course, with temporary exacerbations, attacking the individual four or five days after his arrival in Cochin China. The symptoms are: loss of appetite, chilliness, and general malaise; the tongue also is coated, there are colicky pains, nausea, sometimes vomiting and increased rapidity of the pulse. On the twelfth to the fifteenth day, the patients awake with rumbling in the bowels and desire to defecate; the fæces are clay-colored at first, but later are fetid and have a peculiar stale smell; the same symptoms are repeated nearly every morning. After a long siege of the disease, there are active disturbances of digestion and its consequences, well-marked anæmia and loss of strength; the skin is pale, dry, wrinkled, and grayish-brown; the eyes are dull and voice is affected; there is great sensitiveness to slight draughts of air, also stomatitis. The alvine discharges are then very abundant, but there is no admixture of blood. Hunger and thirst are also marked. The temperature is usually normal or under the normal, seldom elevated; the pulse is apt to be more frequent, and the passages are increased so that twenty to thirty are seen within the twenty-four hours. The minds of the patients, even up to the fatal ending, are clear; recovery in Cochin China is hardly known, nor does a voyage to Europe save them, for ninety per cent of those who attempt to return die.

The disease lasts from two or three months to several years. The post-mortem appearances have little that is characteristic. The mucous membrane of the small intestine is colored brown or gray, and injected in some instances; in others it is white and appears as if it had been macerated in water; sometimes it is softened and thickened, while the secreting elements are atrophied or œdematous. There are also ecchymoses in the mucous membrane of the small intestine, sometimes accompanied by superficial erosions the size of a pin's head; the liver is usually atrophied, while parts have undergone fatty degeneration; the pancreas and spleen are atrophic; in the blood, there is free pigment, causing the peculiar coloration of the skin.

In mild cases of the disease, a milk diet is to be recommended; in severe ones, it is useless and will not be borne continuously. Active parasiticides should certainly be tried, such as oil of turpentine, benzine, etc. A strengthening diet is also desirable in order to sustain the vital powers. *Normand* has given notice that he has made experiments with anthelmintics, and we may, therefore, soon hear something from him on this topic.

GENERAL DIAGNOSIS AND THERAPEUTICS

OF THE

DISEASES OF THE LARYNX.

BY

GEORGE M. LEFFERTS, M.D.,

OF NEW YORK,

CLIN. PROF. OF LARYNGOSCOPY AND DISEASES OF THE THROAT, COLLEGE OF PHYSICIANS AND SURGEONS,
NEW YORK; CONSULTING LARYNGOSCOPIC SURGEON TO ST. LUKE'S HOSPITAL, ETC.

GENERAL DIAGNOSIS AND THERAPEUTICS.

BIBLIOGRAPHY.

General Literature.

Burow: Laryngoscopischer Atlas, mit Text. Stuttgart, 1877.—Isambert: Conférences Cliniques sur les Maladies du Larynx. Paris, 1877.—Rauchfuss: Die Krankheiten des Kehlkopfes im Kindesalter. Gerhardt's Handbuch, Bd. III., 1878.—Browne: The Throat and its Diseases. London, 1878.—Weinberg: Size of the Larynx at different Ages. Archiv für Klin. Chir., Bd. XXI., Heft 2d, 1877.—Koch: On Concussion of the Larynx. Ann. de l'Or. et du Lar., Sept. 1st, 1877.—Gavarret: Acoustique Bibliogique. Physical Phenomena of Phonation. Masson. Paris, 1877, in -8.—Hornus: Difficulties of Speech. Thèse de Paris, No. 272. Delahaye, Paris, 1877, in -8.—Fournié: Physiol. of Voice, Delahaye, Paris, 1877, in -8.—Illingworth: Human Voice. Edin. Med. Jour., Vol. XXII., p. 541, and in Nov. No., 1877, p. 427. — Chaumel: Laryngeal Complications in Typhoid Fever. Thèse de Paris, No. 324, 1877.—Sidlo: Simulated Loss of Voice. Significance for Military and Judicial Surgeons. Wiener med. Presse, No. 50, 1877.—Roth: The Epiglottis and Glottis in the Embryo. Centralblatt für med. Wissensch., No. 1, 1878.—Krieg: A Case of Spontaneous Fistula Laryngis. Virchow's Archiv, Jan. 2d, 1878.—McSherry: Laryngeal Stenosis. Maryland Med. Jour., Jan., 1878.—Cohen: Incarceration of Epiglottis, producing Suffocation in Laryngismus Stridulus. Frankel, Waldeburg et al.: Stenosis of the Glottis. Wiener med. Presse, April 21st, 1878.—Koch: Laryngeal Stenosis after Typhoid Fever. Ann. de l'Or. et du Larynx, May 1st, 1878.—Oertel: Formation of Registers in the Human Voice. (Laryngostroboscopy.) Centralblatt für med. Wissensch., Nos. 5 and 6, 1878.—Oertel: New Mode of observing Vibrations of the Vocal Cords. (Trans.) Med. Times and Gazette, Mar. 30th, 1878.—Panas: Injuries of the Larynx—Five Cases. Ann. de l'Or. et du Larynx, March 1st, 1878.—Sommerbrodt: New Form of Submucous Laryngeal Hemorrhage. London Med. Rec., May 15th, 1878.—Zaverthal: Saggi Clinici dei Chirurgia Laryngologica. Roma, 1877.—Fessard: Fracture of Laryngeal Cartilages. Thèse de Paris, No. 522, 1877.—Koch: Laryngeal Stenosis after Typhoid Fever. Art. II. Ann. de l'Or. et du Larynx, July 1st, 1878.—Oertel: Laryngological Instruction. Brochure in 8vo. Vogel, Leipzig, 1878.—Schroetter: The Treatment of Laryngeal Stenosis. Imp. Med. Soc. Vienna, Lond. Med. Rec., June 15th, 1878.—Comandré: On Aphonia. Lyon Méd., July 14th, 1878.—Follenfaut: Nervous Aphonia. Thèse de Paris, No. 53, 1878.—Margary: Chirurgica Laryngoscopica. Gaz. delle Cliniche, No. 3, 1878.—Elsberg: The Larynx in Cases of Leprosy. N. Y. Med. Rec., Jan. 4th, 1879.—Fernet: On Herpes of the Larynx. La France Méd., No. 93, 1878.—Ganghofner: The Anomalies and Narrowings of the upper Air Passages. Vierteljahressch. für pract. Heilkunde, Bd. 141, 1879.—Hack: The Mechanical Treatment of Laryngeal Stenosis. Volkmann's Sammlung klin. Vortäge, No. 152.—Heymann: Phonatory Spasm of the Ventricular Bands. Berliner klin. Wochensch., No. 40, 1878.—Menzel:

Chronic Laryngeal Stenosis. Resoc. san. di Osp. di Trieste, IV., 1878. Ory: Formulæ in Cases of Hoarseness and of Aphonia. Jour. des Sci. méd. de Louvain, No. 22, Vol. II., 1878.—Pieniaczek: Researches upon the Sensibility of the Laryngeal Mucous Membrane. Stricker med. Jahrbücher, Heft IV., 1878.—Seiler: Gangrene of the Epiglottis. Trans. Path. Soc., Phila., VII., 1878.—Beschorner: On the Pathology of the Voice. Jahresb. d. Gesellsch. für Nat. u. Heilkunde in Dresden. Leipzig, 1879, 127-148.—Janicot: Contribution to the Study of Laryngeal Abscess. Thèse de Paris, March, 1879.—Cohen: The Throat and Voice. Phila., 1879.—Cutter: The Uterus and the Larynx. St. Louis Med. and Surg. Jour., Nov., 1879.—Friedreich: On Percussion of Larynx and Trachea. Deut. Archiv für klin. Med., Sept. 23d, 1879.—Lomikowsky: The Laryngoscopic Appearances in Sclerosis and Cerebro-Spinal Plaques. Berlin. med. Wochensch., No. 41, 1879.—Massei: Chorea Laryngealis. Ann. de l'Or. et du Larynx, No. 5, 1879.—Tshiltof: Practical Observations on Laryngeal Surgery. Vilna, 8vo, 1879.—Stöerk: Two Cases of Luxation of the Left Arytenoid Cartilage. Wiener med. Wochensch., No. 50, 1878.

Laryngoscopy.

Hirschberg: Virchow Archiv, 69, S. 146.—Löri: Pesther Med. Chir. Presse, No. 25, 1874.—Unna: Monatssch. für Ohrenheilkunde, No. 4, 1878.—Bose: Deut. Klinik, No. 15, 1866.—Siegle: Berlin. klin. Wochensch., No. 23, 1874.—Böcker: Deut. med. Wochensch., 1876, Nos. 34, 35.—Berthold: Berlin. klin. Wochensch., No. 25, 1875.—Fraenkel: Deut. med. Wochensch., Nos. 41, 51, 1876.—Lucae: Berlin. klin. Wochensch., S. 432, 1875.—Fraenkel: Berlin. klin. Wochensch., No. 16, 1876. (All of the above references relate to the use of various mirrors, combinations of glasses, methods, and illuminating apparatus.) Hirschberg and Fraenkel: Rectification of Laryngoscopic Picture. Berlin. klin. Wochensch., No. 4, 1877.—Cadier: A new Laryngoscope. Gaz. des Hôp., Mar. 2d, 1878.—Unna: New Laryngoscope for Examination of Post. Laryngeal Wall. Monatssch. für Ohrenheilk., Apr., 1878.—Jurasza: Laryngoscopy. Crakow, 1878.

Rhinoscopy.

Michael: Krankheiten der Nasenhöhle. Berlin. S. 6, 1876.—Duplay: Traité élement. de Pathol. externe par Follin. Paris, Tome III., p. 748.—Troeltsch: Ohrenheilkunde. Leipzig, S. 317, 1877.—Michael: Berlin. klin. Wochensch., 1873, No. 34; 1875, No. 41.—Zaufal: Archiv für Ohrenheilkunde, Bd. IX., S. 133-138; Bd. XI., S. 52-60.—Lucae: Virchow Archiv, Bd. 64, S. 476.—Zaufal: Anterior Rhinoscopy. Archiv für Ohrenheilkunde, Bd. XXII., Heft 4th, Sept. 7th, 1877.—Baginsky: The Rhinoscopic Methods of Examination and Operation. Volkmann's klinische Vorträge, No. 160, 1879.

Rhino-Pharyngoscopy.

Zaufal: Archiv für Ohrenheilkunde, Bd. XII., S. 244.

Pharyno-Rhinoscopy.

Schalle: Arch. für Ohrenheilk., X., S. 149.—Krishaber: Ann. des Mal. de l'Oreille et du Larynx, Tome I., p. 42, 144.—Poinsot: Jaccoud. Dictionnaire, Tome 24, p. 25.

Use of the Sensitive Flame.

Gerhardt: Deut. Archiv für klin. Med., Bd. XVI.—Tobold: Deut. med. Wochensch., 1878.—Fraenkel: Deut. Zeitsch. für pract. Med., No. 52, 1878.—Klemm: Archiv für Heilkunde, Bd. XVII., S. 516, 1876.—Tobold: Manometric Flame in Diagnosis of Unilateral Paralysis of Vocal Cords. Deut. Med. Wochensch., Apr. 20th, 1878.—Fraenkel: The Application of the Sensitive Flame to the Diagnosis of Diseases of the Vocal Organ. Berlin. klin. Wochensch., No. 51, 1878.

GENERAL THERAPEUTICS.

OERTEL: Deut. Archiv für klin. Med., Bd. XV., S. 257.—ZÜLZER: Bericht der Schlesischen Gesell., 1861.—SEMELDER: Gaz. des Hôp., No. 134, 1863.—TAUBE: Deut. Zeitsch. für pract. Med., S. 422, 1878.—MICHAEL: Deut. Zeitsch. für Chir., Bd. II., S. 154.—CORSON: Borax and Nitrate of Potash in Hoarseness. La France Méd., Oct. 27th, 1877.—EYSELEIN: Jaborandi and Hoarseness. London Pract., Feb., 1878.—SCHECH: Galvano-Cautery in Laryngeal Surgery. Aerztl. Intelligenzblatt, Nos. 43, 44, 1877.—SMITH: Clinical Observations on Reflex Cough. Am. Jour. Med. Sc., Apr., 1878.—PEARSON: The local Application of Ext. Conii Fluid. in some Diseased Conditions of the Throat. Maryland Med. Jour., IV., 225, 1879. —SEILER: Hand-book of Diagnosis and Treatment of Diseases of the Throat and Nasal Cavities. Phila., Lea, 1879.—SCHAFFER: Treatment of Laryngeal Stenosis due to Perichondritis Laryngea. Deut. med. Wochensch., No. 9, 1879. —ZAVERTHAL: Local Anæsthesia in Endo-Laryngeal Operations. Riv. Chir. di Bologna, 2, S. IX., 16-21, 1879.—MOSLER: Sea Water in the Treatment of Chronic Catarrh of the Throat. Berlin. klin. Wochensch., June 2d, 1879.—RUMBOLD: On Applications to the Pharynx. St. Louis Med. and Surg. Jour., Apr., 1879.—JAFFRAY: Bromide of Potassium in Spasm of the Glottis. Révue Mens. de Méd. et de Chir., Oct. 10th, 1879.

The combined ingenuity of specialists expended upon many and varied forms of apparatus; the work of *Scheff, Oertel, Cadier, Unna, Navratil, Schalle, Delstanche, Frey,* and others has in reality done but little in recent times to improve our means of inspection and diagnosis in diseases of the upper air-passages. New mirrors, new lamps, new means of making applications are not to-day wanting in the specialty; and the list is constantly increasing. But still the practical worker clings to the principles early laid down in the history of the art by *Türck,* and amplified by the mechanical genius of *Czermak.* They—the faucial mirror, concave-forehead reflector, and artificial illumination—have been thoroughly tried by the test of experience, been found all-sufficient, and being the simplest, are justly regarded as the best means. The newer modifications then, interesting as they are in many instances, must be viewed in the light of mechanical or optical curiosities, or rather mere physiological toys, and from a utilitarian aspect, at least, as of little practical importance. Of more value is the aid rendered by laryngostroboscopy, as a diagnostic means, in affections of the vocal cords. As is well known, the actual vibration of the vocal cords during the production of sounds has hitherto eluded direct inspection. According, however, to *Oertel,* their observation is to-day a matter of little difficulty, and is likely to afford instructive information regarding the physiology of the voice. It is only necessary to employ a light sufficiently strong, and to provide an arrangement by which it shall be rapidly interrupted, to render the vibrations visible. The effect of the interruption of the light is to prevent its impression on the retina from being modified before it can be perceived. Thus it is possible not merely to observe accurately the vibrations of one of the moving cords, but also to compare the vibrations of one with those of the other. The practical deduction is obvious.

The manometric flame of *König* has likewise been found to serve a double purpose in diagnosing affections of the vocal apparatus. *Ger-*

hardt, and later *Tobold*, have with it physically demonstrated vocal fremitus by means of a speaking-tube placed over the cricoid cartilage, while to differentiate the conditions of either side or to recognize a paralysis, it is only necessary, as shown by *v. Ziemssen*, to provide two tubes of equal size, two sensitive flames, and to place the tubes at an equal and corresponding level over the cricoid cartilage. The vocal fremitus obtained in this way, and shown or rendered apparent by means of the flame, presents a true vocal picture. Regular and beautiful flames are only formed if clear and musical voices are used, while hoarse or aphonic voices produce manifold changes in the picture. Hoarseness or aphonia are, therefore, readily appreciated by the use of the apparatus, but whether or no it will ever come, in its application, into a general or practical use remains to be seen. *Fraenkel* does not admit that it is of use for diagnosing different degrees of aphonia, because the apparatus is too sensitive and subject to too many influences. *Klemm* is more enthusiastic and claims to establish with its aid the most minute variations from the true vocal picture. As a matter of fact, with it unquestionably can vocal differences be detected, grades of hoarseness recognized, and physiological information of a valuable nature be obtained; but the trained ear cannot be undervalued through its rivalry or superseded in the certainty of its appreciation in differences of sound. With *Fraenkel* we believe that it will only be used with success by those who are unfortunately deaf laryngoscopists. As a further means of certainty in diagnosis, *Friedreich* extols the utility of laryngo-tracheal percussion, and claims that it possesses a scientific interest, unaltered by the introduction of the laryngoscope with its visual certainty and precision. By means of percussion the demonstration of pure physical facts, in an evident manner, is possible, and different processes in the interior of the larynx carried to an acoustic expression.

Aside from these points, moreover, the careful review of the results of such percussion will lead to the appreciation of diseased conditions of the parenchyma of the lung.

Laryngoscopists will, however, hardly agree with him that the practical results of such percussion will at all compare with those furnished by the laryngeal mirror.

While the means of observation have thus been optically and physiologically amplified, more perhaps theoretically than practically, as regards the larynx, and while the direct mechanical aids have shown no practical or vital improvement within very recent times, rhinoscopy, and through its means our knowledge of pathological change, and our skill in operations in the nares and naso-pharyngeal space has been greatly extended and improved. This fact is well shown in the added sections on Rhino-pharyngoscopy and Pharyngo-rhinoscopy in the revised edition of *v. Ziemssen*, and the well-known labors of *Voltolini*, *Zaufal*, and others, th value of whose works, especially that of the former, is well recognized b all specialists.

Therapeutics are notoriously fickle; a means to-day extolled is to-morro condemned. Constant change is the order of the day, and but few remedies survive the test of prolonged experience. Laryngeal medication forms no exception to the rule, but the fault, if fault it be, exists to a lesser degree, it can be fairly claimed, than in other departments whose armory of medicaments is more extensive.

The laryngeal insufflator, spray, brush, and probe, the astringent, alterative, or caustic solution still play their old rôle. In local laryngeal therapeutics the changes of modern times consist not so much in the experimentation with new remedies, as in combinations and changes of indication in the use of the old. The use of the cautery-iron has won many ardent supporters, among them *Michel.* With the galvano-cautery or thermo-cautery of *Paquelin,* used in the pharynx, nares, or more rarely in the larynx, do we to-day destroy or powerfully constringe the parts, and unquestionably attain results heretofore impossible. A long step in advance has thus been made.

INFLAMMATION OF THE LARYNGEAL MUCOUS MEMBRANE.

BIBLIOGRAPHY.

Staffella: Wochenblatt d. Ges. d. Wiener Aerzte, S. 154, 1862.—Gerhardt: The Catarrh of Measles. Jenaische Zeitsch., Bd. III., 118.—Klemm: Jahrbuch für Kin. Heilkunde, n. F., Bd. VIII., S. 360. Cases of Laryngitis hypoglottica chron. hypertroph. See v. Ziemssen, 2d ed., pp. 230–231. Treatment. Schrötter. Catti. Ganghofner. v. Ziemssen, 2d ed., p. 239.—Stoerk: Klinik der Krankheiten des Kehlkopfes. Stuttgart, 1880.—v. Ziemssen: Handbuch, Vol. IV., 333.—Dittrich: Prager Vierteljahrsch., 1850, Vol. 27.—Taeter and Krishaber: Treatment of Œdematous Laryngitis. Med. and Surg. Reporter, Dec. 1st, 1877.—Fessard: Fractures of Laryngeal Cartilages. Thèse de Paris, Dec. 15th, 1877.—Michel: Angina Epiglottica Anterior. Centralblatt für med. Wissensch., No. 2, 1878.—Berdinel: Œdema of the Glottis. Gaz. méd. de Paris, Fev. 2, 1878.—Whipham: On Treatment of Acute Laryngitis. Med. Times and Gaz., April 20th, 1878.—Catti: Etiology and Treatment of Chorditis Vocalis Inferior Hypertrophica. All. Wiener med. Zeitung, Nos. 25, 26, 30, 1878.—Gauché: Glottic and Subglottic Laryngitis, *à propos* of Erysipelas of the Pharynx, Face, etc. Bull. Soc. Anat., p. 589, 1877.—Stoerk: Acute Hypertrophy of the Laryngeal Mucous Membrane. Wiener med. Wochensch., July 13th, 1878.—Baginsky: The Laryngoscopic Appearances in Croup. Central-Zeitung für Kinderheilkunde, No. 1, 1878.—Schroetter: On Chorditis Vocalis Hypertrophica. Monatssch. für Ohrenh., XII., 1878.—Catti: The Causation and Treatment of Chorditis Vocalis Inferior Hyper. Schmidt's Jahrbuch, Bd. 181, No. 3, 1879.—Bary: Acute Laryngeal Œdema the first Symptom of a Nephritis Scarlatinosa. Central-Zeit. für Kinderheilk., No. 19, 1879.—Bourdon: Suffocative Laryngitis occurring during Convalescence from Scarlatina.—Gaz. des Hôp., No. 89, 1879.—Meyer: Phlyctenular Inflammation of the Vocal Cord. Berlin. klin. Wochensch., No. 41, 1879.

Among the occasional dangerous results of an acute inflammation of the laryngeal mucous membrane, the attention of observers has been called in recent times to the sudden and acute swelling of the tissues immediately below the vocal cord and in the lower cavity of the larynx, with urgent and rapid stenosis (laryngitis hypoglottica acuta gravis). We deal here with a region which until recently was always supposed to be the seat alone, if morbidly affected, of chronic processes, which might, it is true, lead to stenosis, but always slowly and through the deposit and contraction of plastic material—*chorditis vocalis inferior;* acute œdema having always been, in the literature certainly, ascribed to acute inflammatory changes of the tissues of the upper parts of the larynx. But the laryngoscope and time have worked a revolution of ideas. *Burow, Lefferts,* and *Rauchfuss* have observed and reported cases of acute sub-glottic inflammation with œdema in children, and *v. Ziemssen* in the adult. Their importance from a diagnostic point of view can readily be appreciated. In its symptomatology and even results the affection does not vary from those of croup. In children it has beyond question always been regarded as such; while its unsuspected frequency and its dangers lend to it a peculiar interest. In this connection also, attention may be called to a mechanical factor in infantile spasm of the larynx, *Laryngismus stridulus,* which is certainly not generally recognized. *Cohen* believes that the spasm of laryngismus affects the aryteno-epiglottic muscles, in some instances at least, as well as those muscles which close the glottis; and that incarceration of the epiglottis, its forcible drawing down by their spasmodic action, so that its free edge becomes wedged between the posterior face of the larynx and the wall of the pharynx, continues after relaxation of the spasm, and may be the immediate cause of death as he has seen. In undoubted cases of this kind, tracheotomy may be absolutely indicated as necessary to avert asphyxia in recurring paroxysms of spasm.

Among the newer pathological lesions of the larynx in inflammation of its mucous membrane, and in certain diathetic conditions upon which modern research has enlightened us, stands the *phlyctenular inflammation of the vocal cords,* of *Meyer,* and the *herpetic laryngitis* of *Beregszaszy.* Herpes of the pharynx has been long known and recognized, but few cases of herpes of the larynx are upon record; this rarity may depend on a paucity of investigation. Herpes of the larynx presents no differences either in pathology or ætiology from the like affections in the pharynx; it appears upon the epiglottis, the vocal cords, and the general mucous membrane of the larynx in the form of little exudations, seated upon an inflamed base, which one to two days later change to vesicles, and finally disappear, leaving a small erosion or ulceration, which, in turn, heals over in a few days even without treatment.

The vesicles do not all appear at the same period, and in given cases may be observed in all stages of development, at the time of one examination. Vesicles upon the tongue, lips, and in the pharynx will occur concomitantly. During this process the epiglottis will be more or less swollen and reddened, and the vocal cords, dependent upon the number of the vesicles, markedly or slightly injected or thickened. The subjective symptoms are not marked. It has been shown, moreover, that the differential diagnosis in the affection may become a matter of moment. *Meyer* has seen herpes occur primarily in the larynx, and of such a grade as to render the question as to its true nature no easy one to decide, and tells us that it is readily confounded with ulcerative affections, especially syphilis.

An important point, and one which affords food for reflection, is raised by *Michel* in relation to the part that acute infectious diseases play, in children, in the production of chronic laryngeal affections. Unquestionably in the majority of cases of acute infectious diseases the larynx is more or less implicated. In some the disease is here localized—diphtheria, croup, and whooping-cough; in others several organs participate —measles, scarlet fever, and variola; and in still a third class, typhoid fever, gives rise, under exceptional circumstances, to laryngeal complications. That the local disease may, especially when it occurs in typhoid fever, have a destructive tendency, cause irreparable organic changes, interfere with the functional duties, cause anchylosis and stenosis, is a fact which is well known.

That the local complication in the larynx may persist for a long time after the subsidence of the general disease is not so well understood; and that chronic disease, interfering materially, not only with the voice, but likewise with the general health of the patient, even preventing the *restitutio in integrum* for months, years, or entirely, may remain after the primary affection has made its disappearance. Since *Michel's* attention has been directed to this fact, he has seen a large number of obstinate but uncomplicated chronic laryngeal and tracheal catarrhs as sequelæ of pneumonia, whooping-cough, variola, diphtheria, and the like, and wonders that these diseases are not included in the ætiology of catarrhs of the air-passages in works upon this subject. These simple catarrhs differ in many cases in no respects from other chronic catarrhs. The symptoms are cough, especially in the evenings, and slight hoarseness. Pencillings with iodine in glycerine or tannin cause them to disappear. Exacerbations and recurrences are, however, not infrequent, and are usually caused by exposure. Finally, *Dittrich*, in his excellent essay on perichondritis laryngea, mentions that, in case of abscesses about the plate of the cricoid cartilage which have opened toward the pharynx, an ulcer is very often found on the posterior pharyngeal wall, corresponding accurately to the denuded portion of the cricoid; and toward the end of the essay, when discussing the connection between tuberculosis and perichondritis, he makes the positive assertion that the cause is "decubitus, in consequence of the pressure of the cricoid cartilage, particularly if ossified, upon the soft parts in front of the vertebral column." It is believed that in the typhoid process inflammation of the perichondrium is due to this kind of internal decubitus; *i. e.*, pressure of the plate of the cricoid against the vertebræ. Such an opinion has found expression in the best and latest works, as *v. Ziemssen's*, *Störk's*, etc.; and if it be correct, we must almost necessarily find perichondritis occurring in aged persons, who, through a paralysis or disease of the locomotor organs, are compelled to lie constantly on their backs and have become greatly debilitated. Two hospital cases have recently come under *Gerhardt's* observation which corroborate this view, and illustrate the combination of these factors.

LARYNGEAL PHTHISIS.

BIBLIOGRAPHY.

Doleris: Archives de Physiol., No. 6, 1877.—Frommel: Diss. Würz., 1877. —Fraenkel: Virchow Archiv, Bd. 71, 1877.—Isambert: Paris, 1877 (book).—Kittler: Münchener Diss., 1878. Heinze: Leipzig, 1879 (book).—Seckowiski: Tracheotomy in Tuberculosis of the Larynx. Phila. Med. Times, Oct. 13th, 1877.—Browne: Tubercular Laryngitis. Phil. Med. and Surg. Rep., Jan. 26th, 1878.—Cadier: Laryngeal Phthisis. Treatment of Ulcerations and Œdema of the Larynx by Carbolized Glycerin. Gaz. des Hôp., No. 54, 1878.—Fauvel: Tubercular Ulceration of Larynx. Gaz. des Hôp., Mar. 19th, 1878.—Frommel: Affections of the Larynx in Phthisical Patients. Inaug. Diss. Würzburg, 1877. —Fauvel and André: Alterations of the Larynx in Tuberculosis. The Lancet, Aug. 3d, 1878.—Kittler: Paralytic Disorders of Voice in Phthisis. Aerztliches Intelligenzb., May 28th, 1878.—Pelan: Clinical Study of Laryngeal Phthisis. Thèse de Paris, No. , 1878.—Porter: On Tubercular Laryngitis. Reprint Trans. Missouri State Med. Ass'n, 1878. — Bordenave: On Tubercular Laryngitis. Thèse de Paris, No. 424, 1878.—Pelan: Clinical Study of Laryngeal Phthisis. Thèse de Paris, No. 225, 1878.—Payneau: On Auscultation of the Voice. Thèse de Paris, Aug. 7th, 1878.—Cohen: Laryngeal Phthisis; a Clinical Lecture. N. Y. Med Rec., Dec. 28th, 1879.—Gougenheim: Œdema of the Glottis in Laryngeal Phthisis. Le Mouvement méd., No. 51, 1878.—Naunyn: Methodical Deep Inspiration as a Treatment of Paralysis of the Post. Crico-Arytenoid Muscles. Berlin. klin. Wochensch., No. 47, 1878.—Bordenave: Tuberculous Laryngitis Considered from an Etiological, Anatomical, and Pathological Standpoint; with Symptomatology. Paris, pp. 61, 4to, No. 424.—Bosworth: Laryngeal Phthisis. Part I.: N. Y. Med. Rec., May 17th, 1879. Part II.: idem, May 24th, 1879.—Massei: The Varieties of Tubercular Laryngitis. Comment. Chir. di Pisa, III., 19-26, 1879.—Robinson: Ulcerative Phthisical Laryngitis; its Nature, and the Value of Tracheotomy in its Treatment. Amer. Jour. Med. Sci., Apr., 1879.—Sokolowski: Climatic Treatment of Laryngeal Phthisis. Medycyna. Warzsawa, VII., 97, et seq., 1879.—Creveling: Laryngeal Phthisis. Ohio Med. Rec., Columbus, IV., 156, 1878-9.—Wilson: The Throat in Tuberculosis. Phila. Med. and Surg. Rep., Nov. 8th, 1879.

The vexed question which for years has been argued by many and distinguished observers, as to the local tubercular or non-tubercular nature of the disease called *phthisis of the throat or tubercular laryngitis*, receives, unfortunately, no new light from the information contained within the pages of the latest edition of *v. Ziemssen*. We find there added the views of *Heinze* alone, to those of various authorities with which we are already familiar from our study of the earlier work and the general literature of the subject. As is well known *Laennec's* teaching of the purely tuberculous nature of laryngeal phthisis early met with opposition (*Louis*, *Cruveilhier*, *Trousseau*), and in later times the opposition has gone so far as to deny that tubercle is the foundation of the disease.

On the other hand, the first authorities in pathological anatomy, *Rokitansky*, *Foerster*, and *Virchow*, contend for laryngeal tubercle; in fact the latter recommends the larynx as the most appropriate place to study it.

Heinze, whose opportunities for the study of the question have been unequalled, contends that true tubercle is the sole reason for the occurrence of laryngeal consumption. Phthisis of the larynx without tubercle, he tells us, does not exist.

But still again, on the other hand, ample authority, more recent than this, can be brought to substantiate the assertion that the affection is but a chronic ulcerative laryngitis, existing in a consumptive patient, and modified in its course and results by the tubercular diathesis, one with local lesions, mainly if not solely inflammatory in their character, and wholly unconnected with truly tuberculous changes in the parts. This view, largely held by the younger workers to-day in the field of laryngeal pathology, is certainly borne out by the results of clinical observation, and confirmed by the microscope.

Thus the question as to the true pathology must, with regret, be left still undecided. Individual experience must shape the convictions of each one regarding the truth or falsity of the doctrines taught.

Fortunately it is with this one question alone that they need cope. The other and fully as vital points concerning the symptomatology, laryngoscopic appearances, and treatment of the disease, consumption of the larynx, are, with but few and as a rule unimportant exceptions, fully agreed upon by the authorities of all lands in these recent times. Among these exceptions a question may be briefly mentioned which is at present attracting some attention, and one about the importance of which there can be no doubt. It is the value of tracheotomy as a curative procedure in ulcerative phthisical disease of the larynx. *Robinson*, who with others has studied the problem carefully, and collected a number of instances in which the operation has been performed, holds with reason that, in order that the best attainable results may be secured by tracheotomy, the ulcerative disease of the larynx should not be permitted to make too great progress. In order to have legitimate hopes of benefit to the patient from the operation, the ulcerations must be yet limited in their action to the mucous membrane, or the soft tissues beneath. When the cartilages are attacked by caries or necrosis, and concomitant anchylosis of the articu-

would be almost irrational to expect any very decided

cheotomy, much less an absolute cure. Trache-

ll therefore as a curative procedure, must be performed in the earlier stages of ulcerative disease, or, more definitely perhaps, at that period when the nature of the ulcerations is obvious, and local treatment is of little avail. It may be added that the testimony of all whose experience qualifies them to judge is, that the operation, sometimes curative, is in all proper instances mercifully palliative; and as a means of prolonging life, of relieving suffering, and of meeting ulterior and imperative surgical indications, can with confidence, as to the results be recommended. Further experience will establish its exact position as a therapeutic measure in this dreaded disease.

SYPHILIS OF THE LARYNX.

BIBLIOGRAPHY.

Schech: Deut. Archiv für klin. Med., XX., S. 128, 1877 (Gummata). Deut. Zeitsch. für pract. Med., No. 45, 1877 (Gummata).—**Sechtem**: Wiener med. Presse, 27-31, 1878.—**Klemm**: Syphilis of Epiglottis; Complete or Partial Loss. Archiv für Heilkunde, Dec. 10th, 1877.—**Sommerbrodt**: Narrowing of the Larynx by Membranous Cicatrices after Syphilis. Berlin. klin. Wochensch., Apr. 1st, 1878, and London Med. Rec., May 15th.—**Vierling**: Syphilis of the Trachea and Bronchi. Deut. Archiv für klin. Med., Apr. 16th, 1878.—**Sechtem**: Laryngeal Syphilis. Schnitzler's Poliklinik in Wien. Wiener med. Presse, Nos. 27, 28, 29, 30, 31, 1878.—**Etchebarne**: Syphilis Causing Membranoid Occlusion of the Glottis. Thèse de Paris, Aug. 7th, 1878.—**Krishaber**: Contribution to Study of Respiratory Disturbances in Syphilitic Laryngopathies. Ann. de l'Or. et du Larynx, Sept. 1st, 1878.—**Whistler**: Lectures on Syphilis of the Larynx. Med. Times and Gaz., Sept. 21st, Sept. 28th, Oct. 5th, Nov. 9th, 1878.—**Massucci**: On Local Treatment of the Syphilitic Lesions of the Larynx. Ann. Universali di Med. e Chir., Vol. 246, 1878.—**Settegast**: On Syphilitic Affections of the Larynx. Archiv für klin. Chir., XXIII., No. 267, 1878.—**Whistler**: Lectures on Syphilis of the Larynx. Med. Times and Gaz., Dec. 7th, 1878.—**Mackenzie**: Syphilis of the Larynx. St. Louis Med. and Surg. Jour., Sept., 1879, No. 3.—**Moure**: Laryngeal Syphilis and Phthisis from a Diagnostic Point of View. In 8vo. Delahaye, Paris, 1879.

The secondary and intermediate stages of syphilis of the larynx have been more thoroughly considered than was formerly the case: stages upon which the description of the disease found in modern text-books does not dwell, confining itself mainly to those of a later date, when deep lesion and extensive destruction of the parts confront the observer, and demand at his hands a ready diagnosis and earnest energetic treatment. *Whistler* has, among others, done the profession a service in bringing this matter before them so clearly, and his work merits careful attention. The signs of the disease in the larynx are subdivided as a rule into secondary and tertiary; but as between the two extremes there exists an important class of cases quite distinct from the deeper lesions of the tertiary stage, it seems quite right to adopt the classification of intermediate lesions. The udy of laryngeal syphilis is recent when compared with the investiga- ions that have been made into the manifestations of this disease in other rgans; and there has existed, therefore, naturally enough, until recently, very uncertain opinion as to the lesions usually met with there. This s specially the case in reference to such early lesions as those which follow: [illegible] Catarrhal congestions simulating those arising from ordinary causes. [illegible] Congestions accompanied by diffuse redness and swelling. 3. Mucous atches of various types. More chronic inflammation, occupying, as it were, the period of transition, the signs of which are diffuse redness, hickening, and ragged ulcerations, especially of the vocal cords.

As regards congestions of the larynx in early syphilis simulating ordinary catarrhs, not much need be said. They are for the most part superficial in character, and increased redness is their chief feature.

Another type of laryngeal congestion occurs precisely at the same [illegible]riod as the one described, that of general eruption. In this the redness [illegible] equally diffused over the affected surface like an erysipelatous blush, [illegible] erythema. No small vessels are seen running over the surface. [illegible]gether with this there is swelling, or better perhaps, puffiness, marked [illegible] the epiglottis.

Do mucous patches, or condylomata of some authors, occur in the [illegible]rynx? And if they do, are they at all in proportion to those appearing [illegible]where? Nearly all laryngoscopists testify to having seen them. *[illegible]rck*, *Gerhardt*, *Roth*, *Tobold*, *Zeissl*, *Krishaber*.) *Whistler* believes [illegible] the different views respecting their frequent or rare occurrence [illegible] based upon too limited a number of cases, and that it is also an [illegible]portant consideration how long the case is under observation when [illegible]tempting to decide their relative frequency. He is sure that, though mucous patches are rare when compared with the numberless ones on the mouth and pharynx, still they do occur more often than is allowed by some authors.

An affection of the larynx which he describes is one of chronic inflammation, in which the signs are diffuse redness, thickening, and *ragged ulceration*, especially of the vocal cords. This occupies an intermediate line between the early and later lesions of syphilis in the larynx. It may occur close in the wake of the former, and be the immediate outcome of the catarrhs, and mucous patches of the larynx already described, or it may show itself three or four or more years after the primary sore. In the first instance there would be still remaining as accompanying manifestations some more or less general eruption on the skin, with mucous patches of the mouth, while in the other there may be tubercular eruptions limited to the arms or legs, periosteal inflammations or scars from ulcerating syphilides, with ulcers of the fauces, and chronic glossitis.

The laryngeal ulcers are deep; they are ragged, with thickened edges; they are small, irregular in shape, and often multiple, and the vocal cords upon which they are situated look as though pieces had been torn out of them—still they are comparatively superficial, and are not accompanied by perichondritis, and necrosis of the cartilages as are the burrowing ulcers of a later period. The salient points of distinction between this form of laryngitis and those others with which it is most likely to be confounded, viz., the earliest inflammations of the larynx in syphilis, chronic glandular laryngitis, and specially phthisical laryngitis, are fairly well marked.

The present *status* of the question of syphilis, as it affects the larynx, and as represented by the views of the latest and best authorities, may be summed up as follows: *Czermak*, *Türck*, *Gerhardt*, and *Roth* recognize as early manifestations of syphilis, catarrhs with no very destructive appearances, mucous papules or condylomata, and superficial ulcers; while *Dance* considers the eruptions which he describes—roseolar, papular, and tubercular lesions—to be quite as marked here as on the surface of the body. These are the main features of every description that is found of these early lesions in all subsequent treatises. *Ferra's* observations have led him to oppose the view that definite lesions are found in the larynx corresponding to the chief divisions of the stages of syphilis into primary, secondary, and tertiary. He says that since the laryngeal affection does not correspond to that of the skin in the majority of cases, one is not justified in describing a roseola and the like, implying thereby a direct relation in the order of their appearance in the larynx and on the skin, for no lesion of the larynx, he says, could be foretold by knowing the co-existent one in the skin. He divides syphilitic laryngitis, therefore, into two forms, either of which may occur at any period of the disease; these are non-ulcerated and ulcerated; the former including hyperæmia, œdema, and hypertrophy; the latter comprising the various forms of ulcers, together with their complications, perichondritis, caries, and necrosis. As regards mucous patches, a most typical lesion of secondary syphilis, he considers them quite exceptional, if they occur at all.

These discrepancies in these views must be left to time and continued observation for reconciliation.

The gummy tumor or syphiloma of the laryngeal mucous membrane as such, has not, according to *v. Ziemssen*, been yet sufficiently studied on the living subject, and the fact is evident that their natural history and laryngoscopic appearances find but little place in modern literature. *Schech's* valuable contributions to the subject will then supply a material deficiency. He tells us that the gummata are the rarest of all the many manifestations of syphilis which affect this organ; and belong to the later stages of the disease; that their diagnosis is one of the most difficult in the whole range of laryngeal pathology, the reason lying in the rapidly changing appearances of the laryngeal picture. In the stage of infiltration a certain diagnosis is often impossible. With the syphiloma, gummy tumor, syphilitic tubercle, or syphilitic infiltration of the larynx, all of which terms are indicative of the same lesion, will be found, in the great majority of instances, further specific processes, or at least the sequelæ of earlier lesions, such as cicatrices of the cutaneous surface, or of the mucous membrane, diseases of the bones, or more rarely, of the lymphatic glands; and, in cases where the laryngeal appearances are doubtful, these co-indications of the disease are regarded as so important that, were a gummy tumor or its results present in the skin, periosteum, or mucous membranes, one would unhesitatingly pronounce in favor of the gummatous nature of the laryngeal disease. Though gummy tumors may develop at any point in the larynx, those parts which lie above the level of the glottis appear to be the favorite seat. *Schech* has seen them on the epiglottis, the vocal cords, and on the posterior wall of the larynx. *Mandl* has observed them on the epiglottis and false cords; and *Türck, Nicholas Duranty*, and *Norton*, below the glottis level. They take their origin in the connective tissue, and on those parts contiguous to the bloodvessels, in the mucous membrane, and submucous tissue. Their development is either circumscribed or diffuse, upon which depends their size. Their number will vary as well as their size—we may have a single example, we may have many. To describe the color and the appearance of a laryngeal gummy tumor is a difficult matter, and the reason does not depend so much upon its seat in the superficial or deep tissues, or its diffuse or circumscribed character, as it does upon the stage of its progress in which it is examined; and just here will probably be found the explanation of the diversity of the descriptions that are given by various authors. All are correct, but the observer must remember that each represents the appearance of the tumor at some one particular stage of its development. In gummy tumors of the larynx we may distinguish various stages, as well as in like tumors of other parts. The first, usually accompanied by more or less marked inflammatory reaction, is the stage of infiltration; the second, softening; third, resorption, and fourth, degeneration. To fix accurate limits for these stages, as regards time, is impossible. The symptoms caused by laryngeal gummata vary according to the seat of the affection and the events to which it gives rise. The differential diagnosis from the following affections deserves special attention: Localyzed hypertrophy of the tissues in the posterior commissure of the larynx, of the vocal cords or false cords, met with in chronic laryngitis; papillary outgrowths, which occur in syphilitic persons as a result of chronic catarrh; laryngeal condylomata and abscess; so-called laryngeal follicular bubo—an hypertrophied, degenerated, and suppurating follicle. Finally, the appearance sometimes seen on the free edge of the epiglottis, which is caused by the cartilage showing through the mucous membrane, has been confounded with a gummy tumor in the same locality.

The subject of *hereditary syphilis of the larynx* has, within the past year or two, excited some discussion and much interest, and our knowledge of the question has increased in proportion to the multiplication of reported instances. It is somewhat curious, however, to find, in the second edition of *v. Ziemssen*, the statement, that such cases are not so very rare; while in the first, he mentioned but one, the well-known case of *Fraenkel* in an infant only three days old and supplements it in the second by only four. A careful search through available literature does not substantiate this assertion; and destruction of laryngeal parts in the young, with, as is most common, or without, pharyngeal implication, and due to inherited disease, must still be regarded as among the rarer experiences of the laryngoscopist; *Sechtem* (3), *v. Ziemssen* (1), *Lefferts* (8) have reported cases. *Mackenzie* has never seen a case in a child younger than eleven years. In each of the five examples that he has met with, there was ulceration of the edge of the epiglottis, with exposure of the cartilage. *Rauchfuss* mentions that in the *post-mortem* records of the St. Petersburg and Moscow Foundling Hospital there are a few cases of deep ulceration and perichondritis in infants of from two or three months old; and finally, *Sémon* has seen the larynges of two brothers, both affected by congenital syphilis. There was abundant evidence of syphilis in the parents, and both children, in addition to other syphilitic affections, suffered from laryngeal trouble from the age of one month, dyspnœa being very marked. There was ulceration about the lips, mouth, and pharynx; laryngoscopic examination was very difficult and imperfect. Treatment was conducted irregularly. The elder child died, after exacerbation of the dyspnœa for five days. There was great chronic thickening of the upper aperture of the larynx, with only subordinate ulceration; and it was from the aggravation of this chronic process, rather than from acute œdema, that death ensued. The younger child died in three weeks from acute œdema of the middle compartment of the larynx, which was completely occluded, the upper aperture of the larynx being almost free. Deep lesions of the larynx are very rare in congenital syphilis, and simple chronic thickening of the parts, very rare in adults, had not been observed before in children.

NEOPLASMS WITHIN THE LARYNX.

BIBLIOGRAPHY.

Laryngeal Neoplasms.

Berchoener: Cysts. Berlin. klin. Wochensch., No. 42, 1877.—Böcker: Cystic Fibroma of the Larynx. Berlin. klin. Wochensch., No. 9, p. 269, 1877.—Keller: Cysts of the Epiglottis. Monatssch. für Ohrenheilk., No. 9, 1879.—Iversen: Cancer. Tracheotomy. Hosp. Tidende., IV., 20, 1877.—Bayer : Cancer of Epiglottis. Operation. Ann. de l'Or. et du Lar., Nov. 1st, 1877.—Chapman: Epithelioma Laryngis. Removal. Am. Jour. Med. Sci., January, 1878.—Browne: Cancer in the Throat. Brit. Med. Jour., Feb. 16th, 1878.—Egeberg: Cancer of Larynx. Norsk. Magaz. für Lägerid., R. III., Bd. VI , Fordhande, p. 110.—Marelle: Contribution to the Study of Laryngeal Cancer. Thèse de Paris, Aug. 7th, 1878.—Koch: Primary Laryngeal Cancer. Critical Remarks. Ann. de l'Or. et du Larynx, No. 1, 1879.—Marelle: On Laryngeal Cancer. Bull. gén. de Thérap. April 30th, 1879.—Krishaber: Lessons on Cancer of the Larynx. Ann. de l'Or. et du Larynx, No. 3, 1879.—Krishaber: Cancer of the Larynx. Ann. de l'Or. et du Lar., No. 5, 1879.—Moure: Primitive Cancer of the Larynx. Case, Jour. de Méd. de Bordeaux, IX., 57, 1878-9.—Lefferts: Clinical Account of Cases of Intra-Laryngeal Growths. N. Y. Med. Rec., Feb. 9th, 1878.—Cohen : A Cyst of

the Vocal Cords Removed by Forceps. Phil. Med. and Surg. Rep., Sept. 21st, 1878.—FOULIS: Intra-Laryngeal Growths. Brit. Med. Jour., Nov. 16th, 1878.

LUPUS AND LEPRA OF THE LARYNX.

GROSSMAN: Anzeiger der Gesell. d. Wiener Aerzte, No. 27, 1877.—LEFFERTS: Amer. Jour. Med. Sci., CL., p. 370, April, 1878.—ISAMBERT: 1877, Paris, p. 179. Schmidt's Jahrbuch, Bd. 182, S. 145, 1879.—IDELSON: Berner Diss., 1879.—JURASZ: Deut. med. Wochensch., 1879.—JURASZ: On Lupus of the Larynx. Schmidt Jahrb., Bd. 182, 1879.

OPERATIONS IN THE LARYNX.

SCHECH: Aerztl. Intelligenzblatt, No. 43-44, 1877.—WITTER: Archiv für klin. Chir., Bd. XXI., p. 217, 391, 1877.—BUROW: Sarcoma of Epiglottis. Pharyngotomy subhyoidea. Berlin. klin. Wochensch., No. 8, p. 101, 1877.—REYER: Laryngotomy. St. Petersburger med. Wochensch., 17, 18, 1877.—BRUNS: Laryngotomy for Intra-laryngeal Growths. Hirschwald, Berlin, 1878.—HACK: Endo-Laryngeal Extirpation of a Polypus of the Anterior Commissure. Berlin. klin. Wochensch., No. 10, 1878.—THORNTON: Thyrotomy for the Removal of Laryngeal Growths. Brit. Med. Jour., April 13th, 1878.—VOELKER: Tracheotomy, resulting in Stenosis of Larynx. Deut. Archiv. für klin. Med., April 16th, 1878.—REYHER: Thyrotomy as a Diagnostic Means in Ulceration of the Larynx. Case of Extirpation of the Larynx on account of Carcinoma of the Vocal Cords. Schmidt's Jahrbücher, Bd. 179, No. 9, 1878.—SCHAEFFER: Operation on hard Fibroid Polypus of the Larynx, by the Voltolini method. Monatssch. für Ohrenh., Jan., 1879.—MALINOWSKI: Thyrotomy for the Removal of Intra-Laryngeal Growths. Breslau, 8vo, 1879.—PORTER: Excision of the Epiglottis. Am. Jour. Med. Sci., April, 1879.—SCHROETTER: On the Extirpation of Laryngeal Polypi. Wien. med. Bl., II., 1879.—CATERINOPOULAS: Immediate Thyrotomy in Fractures of the Larynx. Thèse de Paris, August 5th, 1879.—REICHERT: A New Method of raising the Epiglottis in Laryngeal Operations. Hirschwald, Berlin, 1879.—STRAUSS: A New Method of Operating on Laryngeal Polypi. Case. Eira-Göteborg, III., 498, 1878-9.—ZAVERTHAL: Local Anæsthesia in Operations in the Larynx. Ann. de l'Or. et du Larynx, No. 5, 1879.

Some of the more important contributions which have been made within a very recent date to our knowledge concerning the pathology of laryngeal growths are from the pens of *Keller*, *Heinze*, *Elsberg*, and *Jurasz*, while others less elaborate, but still valuable, come from certain specialists well-known in literature, and are substantiated and illustrated by facts derived from their rich and practical experience.

Our restricted space confines us to but a few examples. *Keller*, for instance, deals with the subject of *cysts of the epiglottis*. After alluding to the rarity of large cysts, he raises the interesting question as to the possibility of their having another cause than mere retention of the contents of muciparous follicles. The location of the cyst and its constant recurrence, in a case which he reports, despite treatment, led him to believe that this cause, undoubtedly true for the smaller cysts, will not apply to the larger, which are always located upon the surface of the epiglottis, involving the fossæ glosso-epiglotticæ. *Luschka* describes the hyo-epiglottic membrane as forming a lateral closed duplicature between the greater cornua of the hyoid bone and the edge of the epiglottis with the hyo-thyroid membrane, and says that this duplicature bounds a space laterally, where the anterior wall is made up of the hyo-thyroid membrane, together with the neighboring part of the thyroid cartilage; its posterior, by the epiglottis, and its nearly horizontal roof, by the hyo-epiglottic membrane. This space is filled by fat cells and is rich in blood-vessels, and in it he has found from two to three mucous follicles (bursæ mucosæ) of the size of a pea. He also alludes to the practical importance of the bursæ which lie before and behind the hyoid bone as they often lead to cysts of some size. Their swelling directly under the hyo-epiglottic membrane, in other words beneath the mucous membrane of the so-called glosso-epiglottic ligament, and beneath the fossæ of the same name, naturally tends toward the point where there is the least resistance, and this is the thin membranous roof of the triangle above mentioned. Posteriorly lies the lower part of the epiglottis; while anteriorly and inferiorly, as well as laterally lie the firm hyo-thyroid ligament, and a part of the thyroid cartilage to block the way.

For these reasons *Keller* regards the large epiglottic cysts as hygromata of the above-described bursæ, or little muciparous follicles in the glosso-epiglottic fold, which in their growth involve more or less of the anterior face of the epiglottis, and thus explains the difficulty of emptying the sac, despite frequent incisions, the cause being the fibrous partition walls frequent in hygromata, likewise the rapid reproduction of the contents of the sac, rarely found in epiglottic cysts, and not checked in some instances by energetic cauterizations.

Heinze describes *cysts of the vocal cords* as constituting a typical affection, for the diagnosis of which the following sign suffices, viz., nodular or spindle-shaped thickening of the edge of the vocal band, of the same white tendinous color as that of the normal band. The depression that can easily be produced in the swelling by the point of a probe, serves for the purpose of differential diagnosis between cyst and fibroma; with the following as an additional, and whenever present, characteristic symptom of cyst of the vocal band, viz., that the voice affected by the tumor, suddenly or very rapidly improves, on account of the spontaneous rupture of the cyst.

Finally, in continuation of the subject of pathology, *Jurasz* adds one to the few reported cases of *lupus of the larynx*: *Elsberg* two cases to the equally rare class *leprosy*, and in addition, in an elaborate article, he discusses the subject of *laryngeal papilloma* from a microscopical standpoint, while *Seiler* contributes a valuable series of papers on "Researches in the Minute Anatomy of the Larynx."

In the matter of operations and new operative methods in intra-laryngeal neoplasms there is no lack of novelty. Observers seem in some instances to have become dissatisfied with the relatively older plans of excision and crushing *per vias naturales*, and to have sought out new means of accomplishing the same ends. Whether or no these will stand the test of practical needs is problematical.

Before proceeding to enumerate them, it may be of interest to refer to the experiments to *Pieniaczek*, upon the *sensibility of the laryngeal mucous membrane*, and the deductions arrived at by *Zaverthal* and

Glasgow regarding *local anæsthesia*, points which have a direct bearing upon the use of instruments within the cavity of the organ. The former touched the larynges of nine patients who were blindfolded, by means of various instruments, such as metal and elastic sounds, sponge-holders, pencils, forceps, etc., with the following results: 1. A difference in the temperature of the instrument used was always readily appreciated. 2. Patients were able to discriminate between a hard and a soft body; but could not distinguish a laryngeal brush or sponge as such or these from a hard metallic sound. 3. Rough bodies, such as the pencil holder, the head of the sound, and sponges, were as a rule not to be distinguished by the patient from the metal sound.

Zaverthal reviews the literature of the subject of anæsthesia, giving the experiments tried by *Türck*, *Coen*, *Rühle*, *Tobold*, and *Schrötter*, for producing local insensibility of the larynx by means of chloroform, morphine, bromide of potassium and cold solutions of tannin or alum, and explains the objections of those opposed to this method, such as *Schnitzler, Voltolini, Bruns, Fauvel, Mandl, and Massei.* In order to prove the value of these respective opinions, he gives the result of his own experiments upon dogs, made to test three chief questions:

1. Do anæsthetics applied to the larynx produce complete anæsthesia?
2. If anæsthesia be produced, how long will it last?
3. What dangers or phenomena in general accompany or follow this practice?

He experimented upon large and small dogs with chloroform, ether, morphine, hyoscyamin, distilled cherry-laurel water, and extract of hemlock, with the following results: 1st. Local anæsthesia of the larynx cannot be relied upon, even from the most powerful agents, whatever their nature or the method of their application. When it is obtained, which is rarely the case, it is always accompanied by the constitutional effects of the drug. 2d. Attempts made to produce anæsthesia of the larynx are not without serious dangers resulting from constitutional effects, or severe inflammations. 3d. The dangers vary in degree with the means employed; that of poisoning being very great from the use of solutions of morphine, and if chloroform be used, of severe laryngitis. Of 47 cases, in only 5 was complete anæsthesia obtained. In 27 patients the anæsthesia was incomplete, and the applications had to be repeated two or three times; while in 15 cases it was necessary to abandon it altogether. From this the author decides against local anæsthesia in intra-laryngeal operations, and recommends the following rules: 1st. Disturb the patient as little as possible by preliminary proceedings. 2d. Calculate precisely the curve necessary to be given to the instrument, and arrive *cito et tuto* upon the field of operation. 3d. Prove to the patient that the introduction of an instrument into the larynx will not suffocate him. 4. Plunge the instrument deliberately, gently, but without hesitation to the point to be operated upon. 5. Have a very exact understanding of the case, so as to operate as rapidly as possible.

Glasgow, pursuing the same line of inquiry, arrives at the following conclusions: 1st. Carbolic acid in strong solutions produces anæsthesia and relieves pain. The application causes an intense burning, which lasts about twenty seconds, the anæsthetic condition continues about two hours. 2d. The hydrate of chloral in strong solution applied to the mucous membrane produces anæsthesia. The application causes a severe burning pain, lasting over a minute; the anæsthesia does not continue longer than one half-hour. 3d. The strength of the solution necessary to produce anæsthesia varies somewhat in different persons. 4th. It is recommended that the weaker solution be applied first, and this can be followed by the stronger solution. The first application is the only one causing pain. 5th. No bad results, either constitutional or local, have followed the applications of strong solutions of carbolic acid.

Turning to the operative methods we learn that *Reichert's* new plan of *elevating a depressed or overhanging epiglottis* consists in pressing downward and forward the median glosso-epiglottidean ligament or the tissues in that neighborhood, by means of an instrument shaped at its end something like the handle of the ordinary grooved director; while to accomplish the same end, and obtain a view of the growth, which such an epiglottis so often obscures, *Hack* found that if he caused his patient to take a deep and forced inspiration, and at its height, before expiration commenced, to hold his breath, at this moment the cushion of the epiglottis flattened itself and remained so, and a remarkable anæsthesia of the larynx supervened. In this "breath-pause" before expiration, he could not only see the whole glottis, but for a few seconds introduce instruments into the larynx with precision and safety.

Langenbuch has devised a special operation to which he has given the name, *Laryngotomia subhyoidea vera S. sub-epiglottica.* He reports a case of laryngeal polypus in the anterior commissure of the larynx which was overhung by the epiglottis in such a manner that it could hardly be seen, still less operated upon by the mouth, and which he removed by an operation for which he proposes the above name. It is as follows: Transverse incision through the skin, separation of muscles from the hyoid bone, division transversely just above the upper edge of the thyroid cartilage of the hyo-thyroid membrane; then a vertical incision extending through the ligamentous tissue in the thyroid notch and upper third of the cartilage (though perhaps this last through the thyroid cartilage is unnecessary), and transverse division of the base of the epiglottis. The larynx is now drawn forward and downward with hooks, so that it can be easily looked into. In the above case, the tumor and its point of insertion were easily seen, and as easily removed. Speedy recovery followed.

On the other hand, *Voltolini* manages to remove intra-laryngeal growths without either special instruments or a mirror. He simply swabs out the larynx, and in so doing tears off the soft polypi. The swab consists of a flexible wire to which is attached a sponge, whose diameter does not exceed, at the utmost, one millimetre. The sponge, having been previously softened in water, and then pressed out, is introduced "blindly" into the larynx, which closes upon it. No attempt should then be made to advance or press downward until the larynx opens again. When this takes place, the swab must be passed up and down between the vocal cords. In favorable cases when the sponge is withdrawn the polypi will be torn off. The method is only applicable to comparatively soft growths. It is thought that after the diagnosis has once been made, any practitioner may be able to do the operation successfully. *Voltolini* gives six cases in which this simple method was pursued, and his plan has been followed by *Strauss* and *Schäffer*, who was the first in Germany to remove a soft laryngeal fibroid polypus from the larynx by means of the sponge. The former has likewise succeeded in accomplishing the same result by the same means, so he states, in the case of a hard fibroid growth upon the edge of the right vocal cord, but the growth had previously been cauterized, and small bits removed with the guillotine.

In the matter of novel operations *Rosbach* says that, after many experiments upon animals, he has devised an operation, a new subcutaneous method of removing laryngeal growths, which is as easy as the egg of Columbus, one without difficulty or unpleasant results; one that supersedes the necessity of thyrotomy, or intra-laryngeal means, without pain, and easily and quietly done; an operation which requires no preliminary training of the patient; leaves but a small, punctured wound, needs no after-treatment, and demands only that the physician can use the laryngoscope and that the patient can tolerate the mirror in his throat.

After this enthusiastic commendation, he proceeds to give us the details of his methods. The patient is seated in the ordinary laryngoscopic position, and holds his own tongue—the operator holds the mirror in his left hand or fixes it in an apparatus. In his right he has a small lance, and spear-shaped knife, fixed upon a long shank, which he thrusts below the lower angle of the thyroid notch through the *lamina mediana* of the cartilage in the median line, directly back into the larynx; there is no bleeding. The point of the knife now appears in the larynx at the lower third of the epiglottis, and is given such a direction as to reach the location of the growth, which may now be punctured, incised, or cut away.

The patient experiences no pain aside from that caused in the puncture of the skin; has no sensation of the knife entering the larynx; no cough, and makes no efforts at deglutition. The external puncture is made as a rule 1–5 mm. below the thyroid incisure, so that the point of the knife lies a little above the level of the vocal cords. If we desired to operate below the vocal cords it must be made deeper, even through the crico-thyroid membrane.

The method has been tried upon two patients. In one a cyst lying in the anterior commissure of the larynx beneath the mucous membrane was opened, and in the other a polypoid tumor of the left vocal cord removed at the first sitting. Many and varied experiments have been made upon animals. *Rosbach* believes that this method of operating will be found

LANE LIBRARY. STANFORD UNIVERSITY

efficient, not only in those cases where intra-laryngeal means have failed, and where the patient will not consent to a thyrotomy, but likewise in the majority of laryngeal cases requiring operation, and that here it will be proven superior to the common intra-laryngeal procedures, requiring as it does a shorter time and being much more comfortable for the patient.

It may be said here that the procedure described by *Rosbach* is essentially the same as that devised some time since by *Eyselle* of Halle (the latter passing his needle through the crico-thyroid membrane) and is therefore not new.

Finally, the list of cases in which extirpation of the larynx has been performed for malignant disease and other causes, though augmented in the second edition of *v. Ziemssen*, is still far from complete, and the interesting questions of the indications for the procedure and the after-treatment of the operation itself are dismissed with the most cursory consideration. *Schüller's* excellent article is then most timely, and will be found to contain much valuable and useful information. A brief account of all the operations published or known to the author (nineteen, to which we add the first one performed in America by *Lange*, making in all twenty cases), is given, among which it is pleasant to see a successful one by *Czerny* himself, the originator of the operation. The operation has been done for sarcoma in four cases, for carcinoma in fifteen cases, and for perichrondritis (from unknown cause) in one case. All four of the patients operated on for sarcoma not only survived, but have remained free from recurrence. Of the fifteen from whom the larynx was removed on account of carcinoma, five died of pneumonia and two of exhaustion within two weeks after the operation. Five others died from recurrence of the disease long after the operation.

Two patients (one operated on in September, 1877, by *Wagner;* and one in July, 1878, by *Billroth*) were alive at the time of writing, but there had been a recurrence of disease in the latter. Of one case there had been no late intelligence. The recurrence usually took place, not at the seat of operation, but in the neighboring lymphatics. Of prognosis, in regard to function, *Schüller* says the operation offers everything desirable. It insures not only free respiration through the mouth, but also a restoration of loud voice. It also removes difficulty in deglutition. The prognosis in regard to life and recurrence of the disease depends, as in the case of extirpation of any tumor, upon the nature of the growth and extent of the disease. In our present knowledge, the prognosis is most favorable in cases of sarcoma. It cannot be denied that the operations so far do not offer much encouragement as to the final result in cases of carcinoma, but we must not forget that we are yet in the infancy of the operation, and that the disease was far advanced in most of the cases subjected to it. If extirpation is practised earlier, as soon as the diagnosis is assured, the prognosis will become more favorable. *Schüller* considers that complete removal of the larynx is indicated only in cases of malignant growth. It has been proposed also for organic stricture from inflammatory processes, in order to substitute a larynx which would perform its function for one which would not; but laryngotomy, with partial resection, suffices for these cases, and is far less dangerous; moreover, the preservation of a part even of the laryngeal walls renders the fitting of an artificial larynx easier. If the malignant growth is very circumscribed, removal by laryngotomy may be tried, but, if thorough removal is impossible in this way, extirpation should be done at once. But laryngotomy should not be tried, unless there is good reason to hope for success in this way, because extirpation is rendered more difficult by previous laryngotomy. Extirpation is especially indicated in the large broad-based sarcomata, particularly the soft sarcomata, which cannot be removed laryngoscopically.

NEUROSES OF THE LARYNX.

BIBLIOGRAPHY.

NEUROSES OF THE LARYNX.

Hyperæsthesia.

GANGHOFNER: Prager med. Wochensch., No. 38–40, 1878.—HEGER: Wien. med. Presse, Nos. 14–17, 1877.—CHROBACK: Wien. med. Presse, Nos. 1, 2, 1869.—GRÜNEWALD: Petersburger med. Zeitsch., 1875, p. 575.—ENGELMANN: Hystero-Neuroses of the Pharynx, Larynx, and Bronchi. Rep'nt, Vol. II., "Gynecolog. Trans.," 1878.—MASSEI: Contribution to Study of Laryngeal Neuroses. Il Morgagni, April, 1878.—BEARD: Atonic Voice in Neurasthenia. Jour. Mental and Nervous Diseases, June 15th, 1879.

Anæsthesia.

SCHNITZLER: Wien. med. Presse, Nos. 46–48, 1873.—OTT: Prager med. Wochensch., No. 19, 1877.

PARALYSES.

POYET: Thèse. Paris, 1877.—BÖCKER: Deut. med. Wochensch., Nos. 20–21, 1877.—BRESGEN: Deut. med. Wochensch., Nos. 1, 1877.—GLYNN: Lancet, Sept. 1st, 1877.—SÉMON: Chir. Soc. Trans., London, Vol. XI., 1878.—GERHARDT: Deut. med. Wochensch., No. 4, 1878.—FRAENKEL: Berlin. klin. Wochensch., No. 10, 1878.—MESCHEDE: Berlin. klin. Wochensch., No. 24, 1878.—NAYNN: Berlin. klin. Wochensch., No. 47, 1878.—PIENCAZEK: Wiener med. Blätter, No. 34, 1878.—SCHREIBER: Deut. med. Wochensch., No. 50, 1878.—JURASZ: Deut. med. Wochensch., No. 52, 1878.—SMITH: Amer. Jour. Med. Scien., Vol. 149.—BULL: Norsk. Magaz., 3, R. III., 3, p. 161.—SECRETAN: On Paralysis of the Larynx. Inaug. Thesis, Zurich, 1876.—KOCH: Paralysis of dilator Muscles of Glottis and Laryngeal Paralysis in general. Ann. de l'Or. et du Lar., May 1st, 1878.—SÉMON: Paralysis of Abductor Muscles of Vocal Cords. Lancet, April 27th, 1878.—ROBINSON: Am. Jour. Med. Sci., Vol. 110, 378, 1878.—KOCH: Schmidt Jahrbücher, Bd. 180, 1878.—MASSEI: Ann. des mal. de l'Or. et du Lar., IV., 5, Nov., 1878.—OTT: Prager med. Wochensch., No. 15, 1879.—PEL: Wockblatt von het Nederl. Tijdsch., No. 7, 1879.—JURASZ: Deut. med. Wochensch., No. 14, 1879.—GLYNN: A Case of Paralysis Abductor Muscles. Lancet, Sept. 1st, 1877.—BRIEGER: Complete Paralysis of Vocal Cords, dependent on Degeneration of recurrent Laryngeal Nerves, from

pressure of Tubercular Lymphatic Glands. Berlin. klin. Wochensch., No. 23, 1877.—JOHNSON: Paralysis of one Vocal Cord, from Cystic Tumor of Thyroid. The Doctor, Dec. 1st, 1877.—GERHARDT: Hysterical Paralysis of the Vocal Cords. Deut. med. Wochensch., No. 4, 1878.—MESCHEDE: Bilateral Paralysis of Dilator Muscles of the Glottis. Recovery. Berlin. klin. Wochensch., June 17th, 1878, and Lond. Med. Rec., July 15th.—SMITH: Paralysis of the Abductors of the Vocal Cords. Brit. Med. Jour., July 13th, 1878.—KITTLER: On Paralytic Disorders of the Voice in Phthisis, and two Rare Cases of Paralysis of the Recurrent Nerves, Aerztl. Intell.-Blatt, No. 22, 1878.—LEFFERTS: Two Cases of Bilateral Paralysis of the Dilator Muscles of the Glottis. Cure. N. Y. Med. Jour., Dec., 1878.—SCHREIBER: On Paralysis of the Post. Crico-Arytenoid Muscles. Case. Deut. med. Wochensch., Nos. 50–51, 1878.—SCHREIBER: On Paralysis of the Post. Crico-Arytenoid Muscles. Case. Berlin. klin. Wochensch., No. 42, 1878.—COHEN: Bilateral and Unilateral Paralysis of the Recurrent Laryngeal Nerve. N. Y. Hospital Gaz., March 15th, 1879.—JURASZ: Contributions to the Diagnosis of Phonatory Laryngeal Paralysis. Deut. med. Wochensch., No. 52, 1878.—MORGAN: Dipthonia Paralytica. Nat. Med. Rev., April, 1879.—OTT: On the Cause of Paralysis of the Glottis-openers. Prag. med. Wochensch., IV., 141, 1879.—SÉMON: Tracheotomy in Bilateral Paralysis of the Crico-Arytenoid. Post. The Lancet, May 10th, 1879.—LAZAR: On Bilateral Paralysis of the Abductor Muscles of the Larynx. Breslau, 8vo, 1879. OTT: The Etiology of Paralysis of the Crico-Arytenoid. Post. Muscles. Prag. med. Wochensch., Aug. 23d, 1879 —ARIZA: General Considerations on Laryngeal Paralysis. Case. Anfiteatro Anat., Madrid, VII., 133, 1879.—LE MARCHANT: Laryngeal Paralysis. Paris, 1879, 4to, Thesis, No. 476.—MARTEL: Paralysis of the Crico-Thyroid Muscles. Case. Ann. de l'Or. et du Larynx, No. 5, 1879.—WHEELER: Chorea of the Epiglottis. Bost. Med. and Surg. Jour., No. 16, 1878.—CHARCÔT: On Laryngeal Vertigo. Le Progrès méd., No. 17, 1879.—GASQUET: Laryngeal Vertigo. The Practitioner, August, 1878. —GEISSLER: A Case of Chorea Laryngealis. All. med. Centralzeitung, No. 95, 1878.—MANUEL: Spasm of the Larynx produced by Irritation of the Superior Laryngeal Nerve. All. Wiener med. Zeitung, No. 39, 1878.—JAMES: Stammering of the Vocal Cords. Lancet, Nov. 15th, 1879.—SCHECH: Phonetic Cramp of the Glottis. Centralbl. für med. Wissensch., No. 43, 1879.

In the department of *neuroses of the larynx*, recent time has been fruitful of much elaborate and painstaking work. Indications of the increasing interest shown in the subject will be found in the innumerable cases and papers scattered throughout the literature of all lands; in the increased number of critical examinations; the better understanding of heretofore obscure pathological questions, and in the more satisfactory results of treatment. Glancing at a few of the subjects that have received the latest attention, it is obvious, first, that hyperæsthesia of the larynx has been most carefully studied by *Ganghofner*.

He gives an account of eight cases of purely nervous hyperæsthesia of the pharynx and larynx.

They are mostly such as come often under the notice of specialists, and are a source of much trouble and annoyance both to them and their patients, on account of their pertinacity. In by far the greater number of cases, even the most scrupulous examination fails to detect any anatomical cause by which to explain the troubles; in others, there are minute pathological affections, small erosions in the pharynx, etc., which are, however, too insignificant to account for the sufferings of the patient.

In a few cases, other nervous troubles are observed, such as cardialgia, neuralgic pains, etc., nervous dysphagia, and œsophagismus.

The troubles caused by the disease are: a feeling of burning, pressure, pricking, and dryness in the pharynx or larynx, sometimes in both organs at once; at the same time, the patients complain occasionally of a feeling as if their throat were being forcibly compressed, or as if they had a foreign body in their throat; in some cases, the pain extends as far as the tip of the nose or tongue. If the larynx is affected, spasms of the glottis occasionally ensue, or a purely nervous spasmodic cough without any expectoration; the latter sometimes as often as thirty or forty times daily, but, as a rule, not quite so often.

These phenomena are either persistent, or they appear only periodically, and are then provoked by much speaking, irritating food, or mental emotions. Among the twenty-four cases observed by *Ganghofner* were fifteen female patients and nine males, averaging in age from eight (boy) to fifty-seven years. The etiology of this affection is not clear. It has often been ascribed to anæmia, but anæmia did not exist in every case. It was generally preceded by inflammation of the organs of the throat, simple anginæ, etc.

It often occurs in hysterical patients, but has also been met with in cases where no hysterical symptom was manifest. It has been frequently observed that several individuals of the same family have successively been affected by it, so that there may be an hereditary disposition to the affection. Affections of the genital organs also seem to have some influence on its development.

Ganghofner recognizes two forms of the affection, one due to a continuous irritation of the peripheric terminations of the nerves in the mucous membrane, and another purely central, and not caused by any external influence. In treating the affection, it must always be borne in mind that there is a great tendency to frequent relapses, and that it is a very stubborn disease. The treatment consists in cold baths, sea-bathing, change of air, milk-cures, mountain air, etc., or in the use of the galvanic current, painting the throat with solutions of bromide of potassium, tannin, glycerin, morphine, inhalations of weak solutions of salts, etc. In some cases it will be found advisable to give bromide of potassium, or even to administer hypodermic injections of morphia.

In addition, *hyperæsthesia of the larynx* directly dependent upon reflex uterine causes, or upon manifold remote sources of irritation, as shown in an interesting paper by *Smith*, needs to be mentioned. The first class of cases has been fully elaborated in an admirable article by *Engelmann*, which concerns the hystero-neuroses of the pharynx, larynx, and bronchi; the subject has never been fully worked out, and is one upon which but little can be found in the literature of the specialty.

No better description of the *hystero-neuroses of the pharynx* could be given than that of *Holden*, who has so strikingly verified the dependence of the pharyngeal trouble on uterine derangements. Hystero-neuroses of the larynx are much more frequent than any of the other neuroses. They are shown in the disagreeable and intractable cough which we sometimes find associated with malposition or disease of the uterus.

"The hystero-neurosis of the larynx, a short, hacking, laryngeal cough, will often be noticed in young girls in a mild form, but at times it becomes a source of great annoyance. Examination will, in such cases, reveal a healthy larynx, but generally flexion or stenosis of the uterus, and painful dysmenorrhœa, sometimes uterine cartarrh. The only treatment is *per vaginam*, and as soon as the uterine disorder is improved the cough ceases."

We must not confound with this hystero-neurosis, hysterical aphonia, or the laryngismus of nervous and hysterical women, or those distressing paroxysmal attacks of cough, or of suffocation and strangulation, which are as harmless as they are alarming, and disappear as rapidly as they come.

Cases, either with or without *neurosis of the bronchi*, in addition, are reported by *Hegar*, *Chroback*, *Grünewald*, and *Tripier*. *Cutter* also brings an array of facts and detailed instances to show that organic affections of the laryngological tract do exist in connection with organic uterine diseases, as prime factors, and finally *Smith* carries us into an entirely different field as far as causes go. He treats of *reflex cough*, the primary seat of irritation being outside of the respiratory tract, and in its nature not alone uterine, but manifold. The numerous cases which he cites are full of interest.

Involuntary muscular contraction of the laryngeal muscles, a condition characterized by insufficient force and duration of the tension of the vocal cords in phonation, and owing to a want of co-ordination and persistence in the muscular act, a restlessness of the laryngeal muscles, a twitching contraction of the entire vocal muscular apparatus, is an affection only recently classified by itself, and one to which the name *laryngeal chorea* has been given.

Schrötter tells us that the prominent symptom of the disease is a cough, over which the patient has no control, and which is totally different from that observed in other affections of the air-passages; the cough may be either of a barking, howling, or crowing character. In certain cases of hysteria, such a cough may occur, and it is occasionally simulated, and in many instances it cannot be traced to either of these causes.

Schrötter himself has had eleven cases; they were all young persons, generally from eight to fourteen years of age, in whom, without known exciting cause or other disease, attacks of coughing frequently occurred. The attacks came on every five or ten minutes, when the patient was awake, and disappeared during sleep.

He proposes to call the affection *laryngeal chorea*, because the spasmodic contraction of certain muscles, or groups of muscles, occurs during the waking movements of the patient, and disappears during sleep. Other groups of muscles, in no way connected with phonation and respiration, are occasionally affected. That the affection is a form of chorea, is rendered further probable by the mimicry, the relapses, the course of the disease, and the occurrence of other nervous affections in the same individual, or in members of the family.

Schrötter thinks the affection a motor-neurosis in the strictest sense of the term, but whether it has its origin in the central nervous system, he considers a matter of speculation only. The prognosis is favorable —all of his cases have recovered in from three to six weeks. Relapses occurred, but they were always less severe than the original disease. The treatment consisted in cold shower-baths two or three times a day, the administration of quinine in large doses, and the application of the constant current.

Bell has likewise observed, in a girl of fifteen, a convulsive barking cough, which occurred ten times a minute, and disappeared during sleep. It did not lead to any disease of the larynx. The disease lasted four weeks, and relapsed three times. *Romberg* describes, under convulsive affections of the "nerves of voice," an abnormal tone of voice, which he thought was due to a hysterical or epileptic condition. He reports the case of a woman nineteen years of age, who made such a sound "as a saw made," and so loud that it could be heard on the steps outside the house. He also mentions the case of a physician, sixty years old, who, in consequence of a complicated nervous affection, had occasional attacks of loud bellowing cough. A singular feature in this case was, that soothing influences, and especially music, had the power of preventing the paroxysms, and lessening their violence. *Mandl* describes similar affections. More recently, *Türck* describes similar cases, under the heading of "convulsive coughs," but *Schrötter* thinks it doubtful whether they were due, as *Türck* supposed, to disturbances of innervation about the larynx. In these cases, irresistible paroxysms of coughing occurred, which were of short duration, and of a peculiar barking character. The larynx, trachea, and bronchi were perfectly healthy. *Türck* reports five cases, four of which presented precisely the same symptoms as those reported by *Schrötter*.

Massei has reported three cases which he thought due to hyperæsthesia. His patients were aged respectively, twenty-four, fourteen, and eighteen years, and they presented very slight or no laryngeal disease which could be detected. He considers the affection often incurable. *Geissler* reports the case of a boy, twelve years old, who, after taking cold, had the characteristic cough, which was brought on whenever he attempted to pronounce words or syllables commencing with the letter H.

Subsequently he suffered with general convulsions, with hallucinations and coma, attacks of which could be brought on by pressure or pinching of certain points. And finally, Dr. *Spamer*, in Giessen, has reported a case of the peculiar cough occurring in a child a year and a half old, after an attack of general chorea.

In the same class of cases can perhaps be properly placed the observations of *Schech*, who reports two instances of *phonetic cramp* of the glottis, which he terms dysphonia spastica, as a slight grade of aphonia. In neither case was the voice lost, but partly veiled and partly hesitating—in both cases only momentarily. The laryngoscopic examination presented no noteworthy appearances indicative of the cause of the affection.

Schech believes with *Schnitzler* and *Heymann* that the affection belongs to the same class as writer's cramp, and says that it must be distinguished from chorea of the glottis, from stuttering, and from aphthongia.

Stammering of the vocal cords is the name given by *James*, to the disease which appears to be due to defective co-ordination of the intrinsic laryngeal muscles. The vocal apparatus fails at intervals fully to obey the will, the failure giving rise to sudden interruptions of the voice, while the articulating power may be unaffected.

The sudden interruption of function of the vocal cords is exceedingly difficult to demonstrate. It is in the rapid emission of certain combinations of sounds that the sudden arrest must be watched for. The vocal cords may, with patience, perhaps be observed at the moment when they hesitate or tremble, at a point not sufficiently approximated for phonation, where they may seem to move as with a series of ineffectual efforts to obey the will, or display the irregular, paroxysmal, or spasmodic actions seen in the mouths of stammerers. In less aggravated cases, there may be less distinct interference with voice production, analogous to the defects of utterance called "hesitation of speech."

Finally, the pathology of the disturbance of speech has received an interesting addition through the labors of *Lomikowsky*, who describes the *laryngeal appearances in cerebro-spinal sclerosis*. All authors who describe the disease refer, among other disturbances, to that of speech as a constant symptom, but *Lomikowsky* states that none have as yet carefully investigated this phenomenon, nor made a laryngoscopic examination in such cases. After describing the other symptoms of the affection, which were well marked in his case (man æt. 45), he tells us that the voice was peculiar, a veritable *vox anserina*, during ordinary speech; that a shrill change was remarked in passing from a high to a deep tone; and that the patient was unable, even for a short time, to hold an even note. The cause lay in the impossibility, during the course of a given time, of retaining the tension of the vocal cords equally.

During phonation of the letters *e* and *i*, an irregular vibration of the vocal cords was always seen in the laryngoscope, and was specially noticeable in forcing the tone during the phonation of the letter *i*. The patient was well acquainted with the vocal scale, but was totally unable to take a given note, and felt before such an effort that it would be impossible to succeed. The unequal vibration of the vocal cords (trembling) seen in the case, is ascribed to a defective co-ordination of the muscular power of the laryngeal muscles.

Under the name of *laryngeal crisis*, *Charcôt* describes a very interesting affection attending on or preceding sometimes for several years the development of locomotor ataxy. In criticising this case Dr. *Sémon* says that it is characterized by a feeling of strangulation, and of heat in the larynx, and by a peculiar laryngeal spasm, followed at once by falling down, and by an epileptiform attack. This may repeat itself several times in succession, as soon as the patient has again become conscious. This vertigo is not followed by nausea, and sometimes is limited to the laryngeal spasm, without being followed by the epileptiform attack. Generally the patient falls forward.

The disease is frequently mistaken for cerebral congestion. The entire attack is produced by an irritation of the superior laryngeal nerve (?). *Krishaber* had examined the case which was shown by *Charcôt* as an illustration of the disease, and had found that the glottis was greatly narrowed, much more so than normally. *Sémon* observes that it is much to be regretted that this statement is not precise with regard to the permanency of this laryngoscopic appearance, because this factor is of fundamental importance for the pathology of the entire affection.

If the narrowing of the glottis be permanent in some degree, and this seems to be the case from *Charcôt's* description, it would appear that the posterior crico-arytenoid muscles, which are supplied by the laryngeal recurrent nerve only, must have been involved in the morbid process.

Altogether the symptoms of the case described bear so strong a resemblance to those of an undoubted case of paralysis of these muscles, accompanying locomotor ataxy, shown by him in the Clinical Society of London in April, 1878, and recorded in the society's "Transactions" of the same year, that he cannot help believing that the case shown by the celebrated professor, in illustration of his views on the certainly very rare symptom of locomotor ataxy on which he lectures, was one of not yet far progressed paralysis of the posterior crico-arytenoid muscles, accompanying central disease. *Charcôt* believes that we have no present remedy against these attacks, but thinks they might cease spontaneously.

In the region of the *direct paralysis of the adductor and abductor muscles of the glottis*, there has been no failure of earnestness or success in searching for the truth, in the immediate past. The discussion seems of late to have centred itself upon the ætiology of bilateral paralysis of the posterior crico-arytenoid muscles; certainly a most interesting form of disease, and one requiring prompt treatment to relieve the condition of laryngeal stenosis which it causes. *Koch* attempts to prove that this form of paralysis of the muscles is, in a large proportion of cases, due to central causes; a view upon which there is much divergence of opinion, and one which has excited much interesting discussion, notably at a recent meeting of the Clinical Society of London, where the question whether the laryngeal disease can be due to central disorder, or whether it depends upon local affections, was debated *apropos* of a curious and interesting case presented by *Sémon*. The reader in search of information can be confidently referred to this discussion.

As far as the ætiology of the disease is concerned, this being the present point in dispute, diagnosis, appearances, and therapeutics being to-day generally agreed upon, the cases reported by *v. Ziemssen* in his second edition (the same number as in the first, though many have been published in the intermediate time, which he has not included in his last article) stand as follows: only two of them give any evidence as to the cause of the paralysis, viz., *Riegel's* and *Penzoldt's* cases: in both of them the post-mortem examination showed it to be highly probably that they depended essentially upon a primary neuropathic paralysis, in one instance, of the trunk of the recurrent, in the other of the trunks of the pneumogastric and spinal accessory; while in *Riegel's* second case the muscles were found degenerated, but the nerves intact. The remaining observations contribute but little toward elucidating the pathogenesis. In one instance, the trouble began with catarrhal conditions (*Gerhardt*), in *Rehn's* case it was preceded by typhoid fever, in *Feith's* by erysipelas with secondary pneumonia. In *Mackenzie's* and *Nicholas Duranty's* cases and in *v. Ziemssen's*, the pathogenesis remains entirely obscure.

To this list may now be added many cases (see Bibliography) depending upon hysteria, upon pressure upon the recurrent nerves by foreign body (*Ott*), upon syphilis (*Lefferts*), and upon other and local causes, for a study of which the reader is of necessity, by our lack of space, referred to the original articles.

Finally, as a matter more of curiosity perhaps than of utility, *Naunyn's* treatment of the above affection by methodical deep inspirations may be mentioned. Having observed that the stridulous breathing ceased when the patient made voluntarily some deep inspirations, he availed himself of this discovery by instituting a kind of gymnastic treatment of the affected muscles, consisting in methodical deep inspirations. He states that this treatment was accompanied by perfect success.

In the field of *general surgery of the larynx*, from among many novel and interesting operations, devised as a rule to meet the indications of some peculiar case or condition, and reflecting in many instances much credit upon the ingenuity, skill, and boldness of the operator, we select two cases of *luxation of the left arytenoid cartilage*, reported by *Störk*, in which the treatment adopted was to remove, in one case, a portion of the tissue at the back of the displaced cartilage, and trust to the power of contraction in the subsequent cicatrix to draw it backward into its normal position, or nearly so, so as to enlarge the laryngeal space, which it encroached upon seriously. The attempt was successful. In the second case, the passage of hard rubber-tubes through the larynx for several years was entirely unsuccessful in replacing the cartilage. The position of the vocal cords in both these cases resembled that in double paralysis of the crico-arytenoidei postici.

An operation by *Porter*, in which he removed three-fourths of the epiglottis for neoplasm, is worthy of notice. The main disturbance in such cases seems to be in phonation; the vowel sounds *a* and *e* are less distinct, and the voice harsh, if the cartilage is irregular and jagged. Deglutition, on the other hand, becomes easy after a time, for the base of the tongue may so cover the larynx, and the muscles and mucous folds so close it, that the loss of the organ is largely compensated for. Usually, when ulceration sets in, the process of destruction is so slow that the parts gradually accustom themselves to the loss. After an accident, as when Murat lost a portion of the epiglottis by a musket-ball, it may be necessary to introduce an elastic tube into the stomach as an artificial aid. *Porter* did not find this necessary in his case, which had a history of five months' difficulty in swallowing, with laryngeal pain and cough. On laryngoscopic examination, a large nodule was found occupying three-fourths of the free edge of the organ. After some weeks of local treatment, which accomplished nothing, the diseased mass, including fully one-half of the epiglottis, was severed. Semi-solid food was then ordered, but no artificial aid was resorted to, as the long-continued disease of the part had accustomed the patient to supply its loss. The wound healed in a fortnight, and there was little functional disturbance. The following conclusions are given : If a benign growth of the epiglottis exist, or there is malignant disease which has not as yet implicated the surrounding parts, removal of the epiglottis, or such part of it as is involved, is practicable and justifiable.

The recommendation of *Reyher* is that, in certain cases, especially those of cancer, those in which the disease is located below the vocal cords, those in which a neoplasm is diffuse and ulcerative, in lupus of the larynx and some others, an *explorative thyrotomy* be performed in order that the surgeon may be in a position, not only to make an accurate diagnosis, but to carry out an energetic and effective treatment. The dangers of the operation he considers as trivial compared to its possible results. In a case which he reports, the vocal cords were found to be infiltrated with cancerous deposits. After the above explorative or diagnostic thyrotomy had been made, the entire larynx was then extirpated, according to Billroth's method. The patient died on the eleventh day.

Finally, though it occupies a position perhaps upon the boundary line between general surgery and the special province of the laryngoscopist, attention may be called to an excellent article by *Witte* upon *wounds of the larynx and their treatment*, especially the value of a prophylactic tracheotomy.

DISEASES OF THE SOFT PALATE.

The article on Diseases of the Soft Palate, by Dr. *E. Wagner*, in the second edition of *v. Ziemssen*, is but a reproduction of the article in the first edition, without its being rewritten. No additions that are of any importance can be made to it. An additional and supplementary bibliography of the subject to date is herewith appended:

Calhoun: Defects of Hearing, etc., dependent on hyp. Tonsils. Reprint, Atlanta, 1877.—**Montaz:** On Gangrenous Angina. Lyon méd., July 29th, 1877.—**Piponnier:** On Rheumatismal Angina. Thèse de Paris, 1876.—**Fergus:** Sulphur

and Sulphuric Acid in Angina. Practitioner, p. 341, May, 1877.—Fritzinger: Guaiacum in the Treatment of Quinsy. Phila. Med. and Surg. Rep., March 23 1878.—Petersson: On Angina Phlegmonosa. Upsala. Läkarejören. Forhand XII., 2, 1876.—Sabatier: Fibroma of the Soft Palate. Removal. Cure. Ly méd., No. 16, 1878.—Cornil: Path. Anatomy of Placques Muqueuses of the Tonsi Le Progrès méd., No. 32, 1878.—Mercier: Tumor of the Soft Palate. Spontane Gangrene. Death. Révue mens. de Méd. et du Chir., May, 1878.—Foulis: N on Excision of Tumors of the Palate and Fauces. Brit. Med. Jour., Oct., 12th, 1878.—Hermann: A case of Phlegmonous Angina. Ulceration of the Internal Carotid. La France méd., Oct. 12th, 1878.—Hirschfeld: On Affections of the Tonsil. Wiener med. Presse, No. 41, 1878.—Pinder: On Angina Scorbutica. Wiener med. Wochensch., No. 39, 1878.—Vincent: Adeno-sarcomatous Tumor of the Velum. Lyon méd., April 21st, 1878.—Ehrmann: Phlegmonous Tonsillitis Case. Gaz. méd. de Paris, No. 42, 1878.—Grosholtz: On spreading Quinsey. Brit. Med. Jour., Jan. 11th, 1879.—Potain: Herpetic Angina. Gaz. des Hôp., No. 115, 1878.—Boss: On Deformities of the Velum in Scrofula. Bull. gén. de Thérap., May 15th, 1879.—Broca: Ablation of a Lymph-Adenoma of the Tonsil. Mouvement méd., 1879, XVII., 91.—Fauverteux: A Contribution to the Study of the benign Forms of Scrofulous Angina. Thèse de Paris, No. 263, 1878.—Pierin: On Cancer of the Tonsils. Thèse de Paris, May, 1879.—Verneuil: On Abscess of the Tonsil. Lyon méd., No. 9, 1879.—St. Germain: On Amygdalotomy. La France méd., No. 72, 1879.—Ziem: On certain rare Affections on the Velum. Monatssch. für Ohrenheilkunde, No. 10, 1879.

SPASM OF THE GLOTTIS.

Nothing need be added to Steffen's article on Spasm of the Glottis, except the additional references to bring the matter to date.

Heller: On the Ætiology of Glottis Cramp. Wiener med. Presse, Jan. 27th, 1878.—Tordens: On Spasm of the Glottis. Jour. de Méd. et de Chir., Brux., 1878. —Fleisch: A Contribution to the Ætiology of Glottic Cramp in Children. Deut. Archiv für klin. Med., March 5th, 1879.—Lang: A Contribution to the Pathology and Therapy of Spasm of the Glottis. Cor. Bl. für Schweiz. Aerzte, Basel, IX., 1879.

DISEASES OF THE NOSE.

DISEASES OF THE PHARYNX.

BY

J. SOLIS-COHEN, M.D.,

OF PHILADELPHIA,

PRESIDENT OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION; LECTURER ON LARYNGOSCOPY AND
DISEASES OF THE THROAT AND CHEST IN JEFFERSON MEDICAL COLLEGE; PHYSICIAN TO
THE JEFFERSON MEDICAL COLLEGE HOSPITAL AND TO THE GERMAN
HOSPITAL OF PHILADELPHIA.

DISEASES OF THE NOSE.

BIBLIOGRAPHY.

MAXWELL, G. TROUP: Muriated Tincture of Iron a Cure for Nasal Polypi. Phila. Med. Times, Apl. 17th, 1875.—ADAMS, WM.: On the Treatment of Broken Nose by Forcible Straightening and Mechanical Retentive Apparatus. Brit. Med. Journ., Oct. 2d, 1875.—MICHEL, CARL: Die Krankheiten der Nasenhöhle und des Nasenrachenraumes. Berlin, 1876.—STOERK, CARL: Klinik der Krankheiten der Nase und des Rachens. Stuttgart, 1876.—CASABIANCA: On Thickening of the Septum of the Nasal Fossæ Simulating Malignant Tumor. Journ. de méd. et de chir. prat., Nov., 1876; London Med. Record, Feb. 15th, 1877.—ROBINSON, BEVERLEY: Remarks on Hypertrophy of Turbinated Corpora Cavernosa. Am. Jour. Med. Sci., Apl., 1877.—VAJA, VON: Sarcoma of the Nose in a Syphilitic Subject. Wien. med. Presse, March 18th, 1877; N. Y. Med. Jour., July, 1877.—MACKENZIE, MORELL: Lectures on Diseases of the Nose. The Lancet, July 28th, Aug. 25th, Nov. 10th, 1877.—HARTMANN, A.: The Treatment of Nasal Catarrh. Deutsche med. Woch., Nov. 16th, 1877.—JORDAN, FURNEAUX: The General Utility of Sponges as Surgical Dressings. Brit. Med. Jour., Mch. 30th, 1878.—HARTMANN, A.: Ueber Ozœna. Deutsche Med. Woch., Nov. 13th, 1878.—GOTTSTEIN: Ueber Ozœna und eine einfache Behandlungsmethode derselben. Berlin. klin. Woch., No. 37, 1878.—SCHUSTER and SANGER: On Syphilitic Disease of the Nose. Vierteljahrschrift für Dermatologie und Syphilis, Bd. IV., H. 1 and 2; London Med. Record, Apl. 15th, 1878.—RIEDEL: Tuberculose der Scheidenwand. Deutsche Zeitschrift für Chirurgie, 1878.—COHEN, J. SOLIS: Destruction of an Exostosis in the Nasal Passage by Means of the Burr of the Dental Engine. Med. and Surg. Reporter, Phila., July 13th, 1878.—PORTER, WM.: On Post-Nasal Catarrh. Illinois Med. Recorder, 1878.—VOLTOLINI, RUDOLPH: Die Rhinoskopie und Pharyngoskopie. Breslau, 1879.—SEILER, CARL: Handbook of Diagnosis and Treatment of Diseases of the Throat and Nasal Passages. Phila., 1879.—COHEN, J. SOLIS: Diseases of the Throat and Nasal Passages. 2d Ed., New York, 1879.—HARRISON, REGINALD: Note on the Treatment of Mucous Polypus of the Nose. Brit. Med. Journ., Nov. 15th, 1879.—MILLER, A. G.: The Treatment of Mucous Polypus of the Nose. *Ibid.*, Dec. 13th, 1879.—HARTMANN, A.: Operationen zur Nasenpolypen. Schmidt's Jahrb., Bd. 183, No. 3, 1879.—SPEAR, J. M.: New Method of Plugging the Posterior Nares. Med. and Surg. Reporter, Phila., Nov., 1879.—ALLEN, HARRISON: Errors of Conformation of the Nasal Chambers, Studied in Relation to the Cause of Nasal Disease and Irregularity of the Teeth. Phila. Med. Times, No. 314, 1879.—GOODWILLIE, D. H.: Extirpation of the Bones of the Nose and Mouth by the Use of the Surgical Engine. The Medical Record, N. Y., 1879.—SHURLY, E. L.: The Galvano-Cautery as a Therapeutic Measure in Chronic Nasal and Naso-Pharyngeal Catarrh. St. Louis Med. and Surg. Jour., 1879.—GOODWILLIE, D. H.: Hindrance to the Respiration by Disease in the Nose. Canada Medical Record, Dec., 1879.—ALLEN, HARRISON: On a new Method of Treating Chronic Nasal Catarrh. Am. Jour. Med. Sci., Jan., 1880.—THUDICHUM: Treatment and Complications of Nasal Polypi. The Lancet, Feb. 21st, 1880; Same Author on Polypus and other Morbid Growths in the Nose, their Radical Treatment by the Electro-Caustic Method,

and their Connection with Asthma, *ibid.*, Apl. 17th, 1880.—THORNTON, W. PUGIN: Treatment of Ozœna. Brit. Med. Jour., Mch. 27th, 1880.—HAMILTON: The Surgical Treatment of Epistaxis. Brit. Med. Jour., May 8th, 1880.—WAGNER, CLINTON: A Case of Acute Idiopathic Perichondritis of the Nasal Septum, Terminating in Abscess. Archives of Laryngology, N. Y., Mch., 1880.

STENOSIS.

In re-writing his article on STENOSIS OF THE NASAL PASSAGES, Dr. *B. Fraenkel* alludes to cases such as are related by *Jacoby* (*Arch. f. Ohrenh.*, neue Folge, 6, p. 124), in which stenosis of the osseous framework of the nasal passages has produced such complete obstruction to nasal respiration as to prevent sleep, even under the influence of medicaments; and has thus been the immediate cause of marasmus and death.

CORYZA.

A writer in the *Practitioner* (Jan., 1875) has suggested the prevention of attacks of coryza from draughts of cold air, by wearing cotton-wool in the ears during exposure.

Dr. *Rudolpho Rudolphi* states (*Gaz. med. Ital. Lomb.*, Jan., 1879) that an acute attack of coryza is often relieved in the course of half an hour, by slowly swallowing the saliva secreted while chewing a few dried leaves of *Eucalyptus globulus*. Some reports of this treatment, however, warn against depressing effects of the remedy, even though it stop the cold.

Dr. *Hartmann* recommends compression of the air in the nasal cavities by means of the Politzer air-douche, which drives the secretions out from the adjacent sinuses, and relieves the pains and disagreeable sensations. To prevent any undesirable effects on the middle ear, it is suggested that the external auditory canals be closed by the fingers during the process, to prevent too forcible pressure upon the drum membrane.

The writer may state in this connection that he has sometimes found considerable relief to follow an artificial evaporation of the serous fluids in those cases of acute catarrh which go by the name of "snuffles," and in the acute coryza attacks attendant upon some cases of bronchial asthma, by forcing a current of air through the nasal passages from the ordinary rubber ball and tube, such as is used to propel liquids in spray. The patient carries the little apparatus about him, and retires from time to time to use it as needed.

Porter recommends first relieving the irritation, and then promoting resolution. The patient inhales, several times a day, from a warm, dry goblet, twenty drops of a mixture composed of one drachm of iodine, and ten grains of iodide of potassium, in half an ounce each of ether and chloroform; the vapor from which often gives quick relief. Then he administers five or more grains of carbonate of ammonium every third hour, usually in combination with squill.

CHRONIC CATARRH.

It has become quite common of late years to treat the hypertrophied membrane of the middle and lower turbinated bones in chronic nasal catarrh by the galvano-cautery and galvano-caustic loop, as long ago recommended by *Voltolini*. The loop is used in preference to the cautery whenever it is possible to ensnare a portion of the exuberant tissue; and the cautery in cases where this is impracticable. The eschars left by the process are allowed to fall off before the operation is repeated. Cutting these tissues, where practicable, by the cold loop, as long ago advised by the writer, is likewise coming into considerable use. It is more easy of execution than the use of the galvano-caustic loop, but has the disadvantage of producing hemorrhage, so that the electric method is often actually more time-saving in the long run. It is remarkable, in many instances, how these manipulations are tolerated without causing excessive pain. Occasionally the operations are practically painless. At times they are intensely painful. When there are no means of securing freedom of nasal respiration without the sacrifice of tissue, these procedures are perfectly justifiable. Indeed, they are sometimes essential pre-requisites to the cure of the catarrh.

Gottstein recommends the simple occlusion of the nasal passages by wad-tampon, retained for twenty-four hours in either nasal passage alternately. This prevents desiccation of the secretions, and diminishes fetor by keeping them fluid and thus retarding their decomposition.

Thornton has published a record of six cases of ozæna successfully treated by a nasal spray, applied cold with the hand-ball apparatus, at first daily, and then at longer intervals; and composed of one drachm each of carbonate and biborate of sodium, from half a drachm to two drachms of the chlorinated soda liquor, one ounce of glycerine, and sufficient water to make an eight-ounce mixture.

Evidence is accumulating in favor of the use of iodoform as a local application to diseased conditions of the nasal and naso-pharyngeal mucous membrane, whether ulcerated or not. This remedy is applied in powder, in solution in ether or chloroform, rubbed up with petroleum ointment, and in various other combinations.

Dr. *Allen* makes some pertinent suggestions as to the anatomical construction of the nasal chambers favoring the development of catarrh, when obstructions interfere with the efficiency of their respiratory functions. He places great importance upon the necessity of local surgical interference in overcoming these obstructions or removing them; and has adopted a series of devices for carrying out his procedures in the different portions of the passages; all of which is lucidly described and illustrated in his article.

In syphilitic manifestations, *Schuster* and *Sanger* use the curette or scraping spoon to remove the diseased tissue from the nasal cavity, and thus arrest its progress by contiguity.

NASAL POLYPUS.

A novelty in treatment is extolled by Mr. *Reginald Harrison.* This consists in puncture from the anterior nares by the acupuncture needle, or by *Southey's* trocar, to drain away the serum. This is followed daily, for some time, by thoroughly injecting a solution of carbolic acid and glycerine. Dr. *Miller,* after evacuation by acupuncture, applies rectified spirit in spray, which diminishes the size of the tumor; in many instances, he reports, the polypi have shrivelled up and disappeared altogether.

Dr. *Thudichum,* who has long used the galvano-caustic snare in the removal of nasal polypi, again calls prominent attention to the great value of the procedure, as verified by more than three hundred personal cases.

Dr. *G. Troup Maxwell* recommends the plan first adopted by Dr. *J. H. Reeder,* of Laron, Ill. (*Chicago Med. Jour.*, Sept., 1859), consisting in injecting into the passage a drachm of the tincture of the chloride of iron, with an equal part of water; and kept in contact with the parts for a few seconds by bending the head backward. The procedure is repeated daily. The mucous polypi soon slough, it is said, and are then discharged. The treatment occupies from three to eight days.

EPISTAXIS.

Mr. *Hamilton* advises the following procedure in arrest of EPISTAXIS: "Saturate a piece of linen material, about three feet long and one inch wide, in some astringent;—tea, alum-water, or preferably gallic acid and glycerine. This strip should be regarded as consisting of three parts, each intended for its own special position in the nasal passage. The end of the first portion should be grasped in the dressing forceps and passed along the floor of the passsage and packed into the posterior naris until its presence can be distinctly felt by the finger-tip passed around the soft palate. The second portion should be gradually pressed into the roof of the nasal cavity; while the anterior naris should be filled with the remaining third. Thus every part is thoroughly tamponed. The plug should be left *in situ* until carried away by the natural secretion of the mucous membrane; any part protruding from the nostril being readily cut off with scissors."

Dr. *Spears* suggests as an appliance for carrying a tampon to the posterior naris the use of a flexible gold chain, a short strand of metallic cylindrical beads, or bird-shot compressed upon a cord; the object being that the weight of the appliance shall carry it along the floor of the nasal passage into the pharynx, whence it is drawn into the mouth for the purpose of attaching the tampon in the usual manner.

Mr. *Furneaux Jordan* contends that an ordinary uterine sponge-tent, passed along the floor of the nose, expands into all the cavities of the organ and occludes them effectually.

DISEASES OF THE SEPTUM.

Thickenings of the septum of the nose are occasionally mistaken for tumors and exostoses, deceiving even experienced surgeons into performing unnecessary operations.

Access to intra-nasal osteomas and exostoses is sometimes practicable with the burr and drill of the surgical engine, sufficiently to avoid the necessity for external incisions upon the facial portion of the organ, as exemplified in a case under the writer's care, the mucous membrane and periosteum being first turned off and then replaced after the growth has been ground down. Care is necessary during the manipulation to provide for a stream of water to be kept playing upon the instrument, to keep down the heat from the friction and to wash away the detritus.

Dr. *Clinton Wagner* has described a rare case of acute idiopathic perichondritis of the nasal septum, terminating in abscess with profuse suppuration.

DEFORMITIES.

Dr. *Weir's* method of relieving the deformity of a broken nose consists in an improvement on Mr. *Wm. Adams'* (London) method of refracture and reposition. His article is illustrated by photographs of two successful cases; one in the practice of Dr. *Little.* He performs an osteoplastic operation. *Packard's* bevelled incision is made not more than from one-eighth to one-quarter of an inch in length, over the greatest convexity of the bony deformity and parallel to the free border of the nose, so as to strike, as near as may be, the naso-maxillary junction. Then, by the introduction of a very narrow chisel through this small opening, he cuts through the bone with a few strokes of the mallet. If the tilting action of the imbedded chisel proves insufficient to loosen the other side of the nose, it is only necessary for him to chisel that side also, through the same and only incision. The parts are retained in position by compress and plaster, aided by the use of a nose-truss—a modification of that of *Adams.*

In deformity of the cartilaginous septum impeding respiration, Dr. *Goodwillie* reports success by cutting out the offending portion by means of a pair of gouge forceps, one blade of which is armed with a strong circular knife.

Riedel reports two cases of tuberculosis of the septum narium; one of ulceration of twenty-seven years' standing in a male fifty-three years of age, and the other of ulceration of eighteen months' standing in a female fifty-five years of age. Age and duration are opposed to the hypothesis, though the inference was based on microscopic investigations.

DISEASES OF THE PHARYNX.

BIBLIOGRAPHY.

FOULIS, DAVID: On the Use of the Actual Cautery in Pharyngeal Disease. Brit. Med. Jour., Sep. 22d, 1877.—OSLER: On Miliary Tuberculosis of the Pharynx. Canada Med. and Surg. Jour., 1877.—BROWNE, LENNOX: The Throat and its Diseases. London, 1878.—COHEN, J. SOLIS: Diseases of the Throat and Nasal Passages. 2d Ed., N. Y., 1879.—JAMES, PROSSER: Sore Throat. 4th Ed., London, 1879. SEILER, CARL: Handbook of Diagnosis and Treatment of Diseases of the Throat and Nasal Passages. Phila., 1879.—MACKENZIE, MORELL: Diphtheria, its Nature and Treatment, Varieties and Local Expressions. London, 1879.—LETZERICH: Ueber Natron benzoicum. Berlin. klin. Woch., No. 1, 1879.—STUART, JOHN A. ERSKINE: Note on Sulphur Præcipitatum as a Topical Application in Diphtheria. The Practitioner, London, Oct., 1879.—BURRALL, F. A.: Diphtheria and Benzoate of Soda. The Medical Record, N. Y., 1879.—HOFFMAN, L.: Ueber die therapeutische Wirkung des Natron benzoicum. Berlin. klin. Woch., XVI., 1879.—KROKZAK, F.: Vorläufige Mittheilung über Natron benzoicum Inhalationen am Krankenbette. Wien. med. Presse, 1879.—DABNEY, W. C.: The Topical Uses of Ergot. Am. Jour. Med. Sci., July, 1879.—QUINART: Massage of the Tonsils. Arch. méd. Belges; N. Y. Med. Jour., Aug., 1879.—MACKENZIE, MORELL: A Manual of Diseases of the Throat and Nose. Vol. I., Diseases of the Pharynx, Larynx, and Trachea, London, 1880.—KIEN. A.: De la diphthérie; étude pathogénique et thérapeutique, en particulier de son traitement par le benzoate de soude. Gaz. médicale. Strasbourg, 1880.—JACOBSON, NATHAN: Diagnosis and Treatment of Diphtheria. The Medical Record, N. Y., Mch. 20th, 1880.—BILLINGTON, C. E.: Forty Attested Cases of Diphtheria; with Remarks on Diagnosis and Treatment. *Ibid.*, Mch. 27th, 1880.—ROBINSON, BEVERLEY: An Unusual Case of Extensive Phagedenic Ulceration of the Fauces, without Obvious Efficient Cause. Archives of Laryngology, N. Y., June, 1880. CADY, C. B.: Benzoate of Sodium in Pneumonia (reporting successes in diphtheria). The Medical Record, N. Y., July 3d, 1880.

HYPERTROPHIED TONSILS.

Quinart describes a method of treating hypertrophy of the tonsils, in which he has been quite successful. "He covers his index-finger with alum, introduces it into the mouth, and brings it to bear directly on the tonsil, which is manipulated, with gradually increasing force, over as great an extent of its surface as can be reached. The operation is at first painful and disagreeable, but the discomfort is readily allayed by an emollient gargle. After a few repetitions it ceases to be painful, and the patients readily learn to practise it themselves."

FOLLICULAR PHARYNGITIS.

The opinion is gaining ground that actual destruction of the groups of enlarged follicles, whose existence gives the name to this affection, is absolutely essential to a cure of the disease.

Quite a number of practitioners, on both sides of the Atlantic, engaged in the special treatment of throat affections, express a decided preference for the incandescent galvano-cautery, with which one or more of the offending groups are destroyed daily or at longer intervals, until the pharynx has been cleared. Dr. *Foulis* occasionally prefers, and uses almost exclusively in his clinical practice, the tapering bulb of the ordinary potential cautery, at a dull red or even a black heat. Dr. *Lennox Browne* does not find it necessary to destroy the nodules themselves to effect their obliteration. He simply cuts off their blood supply, by destroying the continuity of the enlarged blood-vessels surrounding them, using the galvano-caustic point for the purpose, and trusts to their subsequent shrinking from want of nutritive fluids.

Dabney extols the free application of ergot, twice daily. He employs a solution of Squibb's solid extract, or ergotine, as it is sometimes called, composed of twenty grains of the extract, one fluid drachm of tincture of iodine, and seven fluid drachms of glycerine.

Dr. *Morell Mackenzie* makes great distinction between the hypertrophic and the exudative forms of follicular pharyngitis, and regulates his treatment of the disease accordingly. When the hypertrophic form exists alone, no remedy is considered by him so productive of good results as London paste, carefully applied to each elevation; not more than two or three being touched on the same day, for fear of exiting too great an amount of collateral inflammation. In the exudative form, his practice is first to scrape the mucous membrane with the curette, wherever the white spots of exudation appear; and having thus cleared the secretion away, to carefully apply a pointed stick of nitrate of silver to each spot which discharges an abnormal secretion.

In applying nitrate of silver to the pharynx and other accessible parts of the throat, the writer has of late years used with great success a simple device of his own which is safe in application, economical of material, and protective to adjacent tissues. The caustic is inclosed in a wooden sheath, in fact, the sheath of an ordinary lead-pencil, like which it is sharpened to a point as required. It can be held in contact with the tissues for several seconds, without breakage, or smearing of contingous surfaces, and is thus rendered effective as a caustic in destroying the offending nodules. It is more easily managed than either London paste or any form of potential cautery.

Stoerck denies the follicular character of these groups of nodules, and declares them to be simply circumscribed masses of swollen epithelial cells denuded of their exterior squamous layer. He removes them bodily with gouge-forceps. Others scrape them off with the curette.

It is the writer's conviction that this destructive treatment is requisite only when it is evident that the nodules excite cough by direct or reflex action, or absolutely keep up the special annoyance or irritation for which the sufferer seeks relief. Many individuals present these enlargements on examination, without ever having been conscious of even uneasy sensations in the throat. The promiscuous use of the galvano-cautery, gouge-forceps, and the like, seems liable to do injury, unless the proper cases are carefully selected by experienced practitioners.

TUBERCULOSIS OF THE PHARYNX.

MILIARY TUBERCULOSIS OF THE PHARYNX, now that it is sought for, is being now and then reported. *Osler* has reported a case of chronic phthisis in which miliary tuberculosis existed in the pharynx as well as in the lungs. Numerous small, firm granulations, which, on examination, proved to be miliary tubercles, were scattered over the posterior and lateral walls of the pharynx. There was no involvement of the larynx, and no ulceration.

DIPHTHERIA.

No new light has been thrown upon the question of the identity of croup with diphtheria.

As regards treatment of diphtheria, Mr. *Stuart* has recently reiterated his encomiums on the topical use of precipitated sulphur, which he now prefers to use rubbed up with a little water and applied with a swab to the desired spot, as easier, safer, and more accurate than any other method.

Dr. *Billington* reasserts his great success with the half-hourly or still more frequent use of teaspoonful doses of a mixture of two scruples of chlorate of potassium in half an ounce of glycerine and two and a half ounces of lime-water. This he frequently, though not invariably, alternates with equal doses of a mixture of one drachm of the tincture of the chloride of iron in an ounce each of glycerine and water. Meanwhile, he sprays the throat frequently with a mixture of ten minims of carbolic acid in four ounces of lime-water. The throat and nasal passages are kept thoroughly cleansed by frequent and efficient syringing with weak, tepid solutions of table-salt.

Great claims of success with benzoate of sodium, in large doses, have been advanced by *Letzerich*, attributed to asserted arrest in the development of the diphtheritic bacteria. These claims have received a certain amount of support from a number of writers, but the weight of rendered evidence is as yet undecisive, to say the least, and a good deal is unfavorable. The formula used by *Letzerich* consists of five grammes of pure benzoate of sodium, dissolved in forty grammes each of distilled water and peppermint water, to which are added ten grammes of syrup of orange-peel. To infants less than one year old, two teaspoonfuls of this are given every hour; to children from one to three years of age, a tablespoonful, the amount of benzoate of soda in the mixture being increased from five to seven or eight grammes; for children from three to seven years old, the proportion of the drug is increased to from eight to ten grammes; for those over seven, om ten to fifteen grammes; and for adults, the proportion is from n to twenty-five grammes. The false membranes are dusted with owder of benzoate of sodium, blown through a quill or other tube, or three times daily in light cases, and every three hours in severe . It is also administered in gargles, a five-per-cent solution being ient for older children.

or removing false membranes bodily from the larynx, when required, *kenzie* recommends an ingenious special device of his own, consisting squirrel-hair brush, the hairs of which are directed upwards. Its ntage for the purpose over the usual form of brush is apparent.

EASES OF THE PLEURA.

BY

G. M. GARLAND, M.D.,

OF BOSTON,

PROF. OF THORACIC DISEASES, UNIVERSITY OF VERMONT.

DISEASES OF THE PLEURA.

BIBLIOGRAPHY.

Ferber, A.: Die physikalischen Symptome der Pleuritis exsudativa, eine klinisch-experimentelle Studie. Akademische Habilitationschrift, Marburg, 1875.—Jobbé-Duval: Thermometrie pleurale. Thèse de Paris, No. 119, 1875.—Rehn, H.: Die wichtigsten Formveränderungen des menschlischen Brustkorbes. Wien, 1875.—Bacelli, G.: Sulla transmissione dei suoni attraverso i liquidi endopleurici di differente natura. Archivio di medicina, chirurgia ed igiene, 1875, fasc. VII., VIII.—Chopinet: Contribution à l'histoire de la pectoriloquie aphone. Union médic., 1876, No. 32.—de Mussy, Gueneau: Etude sur la transmission des sons à travers les liquides endo-pleurétiques. Paris, 1876.—Fea, C.: Etude sur la transmission des bruits respiratoires dans les grands épanchements pleurétiques. Thèse, Paris, 1876.—Hermet, P.: Quelques recherches sur la pectoriloquie aphone. Thèse, Paris, 1876.—Mercadier: De la pectoriloquie aphonique dans la pleurésie. Thèse, Paris, 1876.—Peitavy: Zur radical Operation des empyems. Berl. klin. Wochensch., 1876, No. 19.—Lichtheim, L.: Die Störungen des Lungenkreislaufs und ihr Einfluss auf den Blutdruck. Berlin, 1876.—Oeri, R.: Die Thoracocentese durch Hohlnadelstich und Aspiration bei seröser und eitriger Pleuritis. Stuttgart, 1876.—Peyrot, J. J.: Etude expérimentale et clinique sur le thorax des pleurétiques et sur la pleurotomie. Paris, 1876.—The Same: Sur les tensions intrathoraciques dans les épanchements de la plèvre. Arch. génér., 1876, Juill.—Pfuhl: Ein oberhalb der Leber gelegenes peritonitisches Exsudat in die rechte Lunge perforirt mit den Zeichen eines rechtsseitigen Pneumothorax. Berl. klin. Wochenschr., 1877, No. 5.—Dieulafoy: De la thoracentèse par aspiration dans la pleurésie aiguë. Gaz. Heb., 1877, No. 45.—Fernet, C.: Du déplacement réel et apparent du cœur dans les épanchements pleuraux. France médicale, Feb., 1878, p. 116.—Gleason, J. C.: On Strapping the Affected Side in Cases of Acute Pleuritis. Boston Med. and Surg. Journal, Jan. 24th, 1878.—Powell: On Some Effects of Lung Elasticity in Health and Disease. Med. and Chirurg. Transactions, Vol. LIX.—The Same: On Consumption and Diseases of the Lungs and Pleura. 1878.—Ellis, Calvin: The Line of Dulness in Pleuritic Effusion. Boston Med. and Surg. Jour., Jan. 1st, 1874, p. 13.—The Same: The Curved Line of Pleuritic Effusion. Boston Med. and Surg. Journal, Dec. 14th, 1876.—Garland, G. M.: Some Experiments upon the Curved Line of Dulness in Pleuritic Effusion. Boston Med. and Surgical Journal, Sept. 17th, 1874.—The Same: Pneumono-Dynamics, 1878.—Heitler, M.: Wien. med. Wochenschr., 1878, No. 52.—Rosenbach, O.: Ein Beitrag zur physicalischen Diagnostik der Pleuraexsudate. Berl. klin. Wochenschr., 1878, No. 12.—Stone, W. H.: On Pleuritic Tension. Lancet, Sept. 20th, 1879.—Parrot, M. J.: Les ecchymoses sous-pleurales dans les affections broncho-pulmonaires des enfants. Rev. mens. de méd. et du Chir., 10 Sept., 1879.—Reuss, A.: Beiträge zur klinischen Beurtheilung von Exsudaten u. Transudaten. D. Arch. f. klin. Med., 24 Bd., 6 Heft, 1879.—Weil and Thoma: Zur Pathologie des Hydrothorax und Pneumothorax. Virchow's Archiv, Bd. 75, 1879.—Péan, E.: Contribution à l'étude des déformations des parois thoraciques dans les épanchements pleurétiques. Thèse de Paris, 1879.—Bousseau, I.: Des signes qui permettent de reconnaître l'abondance des épanchements pleurétiques. Thèse de Paris, 1879.—LEYDEN, E.: Ueber Pyopneumothorax subphrenica. Zeitsch. f. klin. Med., Bd. 1, Heft 2, 1879, p. 320.—HOMOLLE: Rév. mens. de méd. et de chir., Paris, 1879, pp. 81-126.—QUINCKE: Ueber den Druck in Transudaten. D. Arch. f. klin. Med., 1878, XXI., p. 453.—ROSER, W.: Zur Operation des Empyems. Berl. klin. Wochen., 1878, No. 46.—WEISSGERBER, P.: Wie entfaltet sich nach der Operation des Empyems die comprimirte Lunge bei offenstehender Pleurahöhle? Berl. klin. Wochenschr., 1879, No. 16.—BOEGEHOLD, E.: Hydrops Adiposus Pleuræ. Berl. klin. Wochenschr., 1878, No. 24.—LISTER, JOSEPH: Clinical Lecture Illustrating Antiseptic Surgery. The Lancet, London, Dec. 20th, 1879.—WEIL, A.: Zur Lehre vom Pneumothorax. D. Arch. f. klin. Med., Bd. 25, Heft 1, 1879.—JAMES, A.: Transudations and Exudations. Med. Times and Gazette, Jan. 3d, 1880.

No advance of importance has been made during the last few years in the investigation of the general or constitutional symptoms of pleurisy. A few new observations have been made upon the temperature of the body during that disease and seem worthy of record. *Peter* reports a case of purulent effusion in the pleural cavity, wherein the temperature of the skin on the diseased side was higher than on the opposite side. *Jobbé-Duval* has found that this difference is a constant phenomenon of pleurisy, even when the effusion is not purulent. *Laboulbene* and *Bonneville* say that the temperature in the rectum rises 0.2°–0.3° C. after aspiration of a pleuritic fluid, but is not affected after puncture for hydrothorax or ascites. These points require further confirmation.

In contrast with the paucity of new general symptoms, we find that our knowledge of the physical signs of pleurisy has been greatly extended by the active work of experimenters and clinical observers. The presence and movements of a fluid in the chest produce certain mechanical results which have hitherto been greatly misunderstood and misinterpreted, but which are now being rapidly brought under the laws of physics and thus placed on a scientific basis. I shall enumerate in order certain of these signs which have been most carefully studied.

Deformity of chest.—Text-books ordinarily speak only of changes which take place in that half of the chest which is occupied by fluid. There are other points, however, in this connection which are of diagnostic value. *Peyrot* injected one side of the chest of several cadavers with plaster-of-Paris, and then made cross sections of the body after the injection had solidified. In this way he discovered that the cartilaginous portions of the ribs on the affected side were bulged forward, thus giving a more rounded appearance to that side, and producing an increase in the distance between the nipple and the middle line of sternum. On the well side, however, the ribs were also modified. They were straightened out so that they appeared flattened more than normal. The sternum was no longer *en face* of the vertebral column, but it was strongly deflected toward the side containing the fluid. In this deflection the lower end travels faster and further (4–5 cm.) than the upper end (2 cm.), so that the whole bone swings like a pendulum as it were. I have recently observed a case of left-sided effusion where this sign was especially well marked. The lower end of the sternum was displaced fully half its width to the left. The deformity of the chest, therefore, is not due to a development of one side, the other remaining normal, but it consists of a mutual adjustment of all parts. *Fernet* thinks that the amount of displacement of heart to the right, with a left-sided effusion, appears greater than it actually is, by reason of the simultaneous migration of the sternum toward the left.

Pneumopericardial friction sounds.—Occasionally fibrinous deposits occur on the outer surface of the pericardium, and on the pleural layer which overlies the same, and produce a friction sound which is coincident with the respiratory movements and also with the cardiac pulsation. This sound may readily be mistaken for an ordinary pericardial friction rub. It may be distinguished by the fact that it is especially prominent along the left border of cardiac dulness, and it diminishes as auscultation is carried toward the sternum and large vessels. There is no enlargement of cardiac dulness.

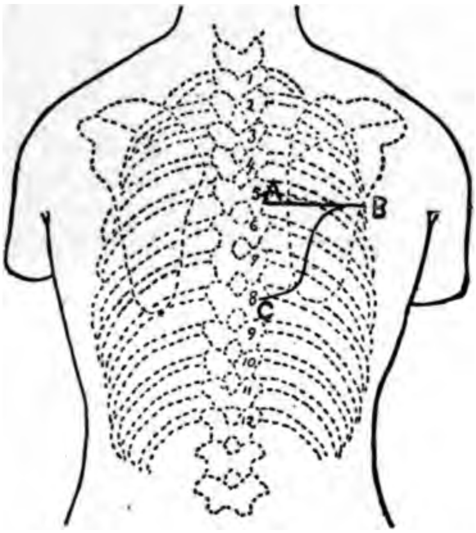
The line of flatness.—The line of demarcation between the fluid flatness of an effusion, and the more or less dull resonance of the lung above, has been variously described by different authors, and great dissension has arisen regarding its true shape and position. Most German writers follow *Wintrich* in declaring that this line stands generally highest behind in the neighborhood of the spinal column, and thence descends obliquely to the sternum. Some allow that the line may sometimes be horizontal, but they think that this shape is exceptional and due to the position maintained by the patient during the early stage of the effusion. Thus, if the patient lie quietly in bed during that stage, the fluid will assume a level corresponding to that position. Subsequently as the patient arises and walks about, the fluid is prevented from reaccommodation by adhesions, and hence the obliquity of its surface. Among the French, *Piorry* and his followers teach that an effusion ordinarily adjusts itself to a horizontal level for all positions of body. On the other hand, *Damoiseau* declared that the line in question is never horizontal, but is more or less parabolic with its summit in the axillary line, and its branches extending down on either side to the sternum and vertebral column.

In my own experience, I have never seen a pleural effusion (pneumohydrothorax excepted) which presented a horizontal line of demarcation, nor do I obtain a line like that described by the Germans. On the contrary, I find that the position assumed by an effusion is that which was first described by Prof. *Calvin Ellis*, of Boston. This observer discovered that with small and medium effusions the line of flatness begins *lowest behind* at the vertebral column. Thence it ascends obliquely across the back, in a *letter* S *curve*, to the axillary region where it reaches its highest point. Then it advances to the sternum with a slight inclination downward. With large effusions, which fill the chest to the second rib or higher, this curve disappears and the line becomes more nearly horizontal and more difficult to trace. As absorption takes place, however, or the fluid is removed by aspiration, the curve reappears and passes through retrograde phases corresponding in shape to those of the earlier stages. In order to discover the explanation of this peculiar shaping of the line, I suspended dogs by the head and injected into the pleural cavity plaster-of-Paris and cocoa butter which subsequently solidified and gave me permanent models of a pleural effusion. When perchance air was admitted with the fluid, and a pneumo-hydrothorax thereby established, the level of the injection was always horizontal, and the air collected between the same and the collapsed lung. When, however, proper care was taken to prevent the entrance of air, a very different condition of things was found. Before opening the chest, I percussed it carefully and obtained a line of flatness which closely resembled the Ellis curve on the human chest. Then on cutting away the ribs I found that the curve corresponded accurately to the line of demarcation between the lower border of the lung and the upper border of the solidified injection.

Several other points of importance were noticed. The lung was smaller in volume, having contracted before the encroaching fluid, but with medium-sized injections it was nowhere compressed or plunged into the fluid as we read in text-books. Even with injections which filled one-half the pleural cavity there was little or no intrusion of the fluid between the lung and chest-wall, but the model all lay between the lung and diaphragm. The lung was not altered in its contour, but the upper surface of the model was accurately adapted to the under surface of the lung. In other words, instead of the fluid compressing and deforming the lung, as we are taught, I found that the lung retained its own outlines, being merely reduced in volume, and it dictated shape to the fluid. Moreover, the entire body of the injection, together with the diaphragm, was held up in the thorax by the lung until the amount of the fluid became excessive. A little reflection taught me that this supremacy of the lungs over the fluid was due to the *retractile* force of those organs. Just as the lung draws up the diaphragm by its retractility during each expiration, so it draws up and suspends the diaphragm plus the fluid. Moreover, by experimenting with rubber balloons in artificial thoraces, with rubber diaphragm, etc., I found that such elastic bodies will not only hold a fluid suspended, but will distribute the same according to the balance of retractility in different parts, *i. e.*, the fluid will adapt itself to those portions of a balloon which retract with most force. I found distributions of the injections within the chest which could be explained in no other way than by differences in the retractile force of various parts of the lung. While I express these mutual adjustments between the lung and fluid, in terms of the retractile body, it will be understood that I recognize the agency of atmospheric pressure and that it is through the action of this agency that the lung is able to move the fluid. The lung acts on the principle of a pump in lifting the diaphragm and fluid, and on the same principle in distributing the fluid according to its own shape.

It will be seen from these experiments that those writers who talk about an effusion presenting a horizontal level have entirely ignored the influence of the lung. Water in an open pail assumes a horizontal level for every position of the pail, but water inclosed in a chest, and subject to the retractile force of a powerful lung, can *not* assume such a level, but must acommodate itself to the shape of the organ acting on it. Of course, when the lung is entirely collapsed and has lost its retractility, it will cease to exert its influence, and the resulting phenomena will vary accordingly. Moreover, pneumonic infiltrations and other conditions which may destroy the elasticity of the lung will also modify the influence of that organ upon an effusion. It will be understood, therefore, that my remarks apply only to the ordinary run of free, non-encysted pleuritic effusions. No law or rule can be laid down for cases which are complicated by adhesions, if these adhesions interfere with the free play of the lung.

It will sometimes be found difficult to trace the curve on the back, owing to the great dulness of the lung immediately above the effusion. This dulness is often due to a lack of proper ventilation of the lower lobe, especially when the patient is lying down, and therefore one should not attempt to trace the line until the patient has taken several deep breaths and thus thoroughly filled the lung. In the diagram it will be seen that I have drawn a horizontal line, A B, from the summit of the curve to the vertebral column, and have thereby inclosed a rough, triangular space, A B C. This space corresponds to the lowest portion of the lung, and is especially liable to be obscured by dulness. The lung lies more in contact with the chest-wall, but its resonance may be so dull as to escape detection unless careful percussion is made and the patient breathes deeply. I have termed this space the *dull triangle*, and its recognition is of vital importance. *Heitler*, in Vienna, has observed this same triangular space of resonance and has likened it to a monk's hood cut longitudinally through the centre and hanging apex down. *Rosenbach*, of Breslau, has also noticed that the resonance of this portion of the back



in pleurisy will often clear up on exercise or by breathing, and such clearing up of the resonance of a dull back he has made distinctive between pleurisy and pneumonia. The same condition of things obtains in hydrothorax, but in some cases the triangle may be still more dull and require careful auscultation and percussion, owing to the œdema of the lung itself.

TREATMENT.

Jaborandi.—The fluid extract of jaborandi has been recommended in pleurisy, and Dr. *Hunt* reports three cases wherein he thinks absorption of the fluid was favored by this drug. He gave it in amounts varying from ʒ i. every four hours, to ʒ iss. every two hours. All the patients bore the medicine well, and one gained in weight while sweating profusely. No inconvenience was experienced except from the diaphoresis and salivation, and no beneficial results were obtained until profuse diaphoresis was established.

Immobilization.—Dr. *J. C. Gleason* recommends a firm strapping of the chest during the painful stage of pneumonia and pleurisy. He cuts strips of adhesive plaster one and one-half inches wide and long enough to reach from spine to sternum. These strips are applied firmly, as in the case of a broken rib, and are said to bring immediate comfort and lessen the necessity for opium.

Thoracentesis.—Very little that is new can be said about the various methods for removing fluid from the chest, but I wish briefly to call attention to the simplicity and comfort of the siphon-method. Take a needle canula, armed with a stop-cock, attach it to a rubber tube about one metre long, and 4 mm. in diameter, and fill the same with a one-per-cent solution of carbolic acid. Plunge the needle into chest; let the other end of the tube hang down into a basin on the floor, open the valve, and the weight of the column of water in the tube will draw off the fluid in a quiet, uniform manner. *Girgensohn* and *Risel* employ a canula which is 2–4 mm. in diameter, and they say that they can empty a chest in fifteen minutes. I believe myself that rapid evacuation is not desirable and may be very dangerous. It seems to me that many of the unfavorable results of thoracentesis which have been recently reported were directly due to the rapidity and force with which the collapsed lung has been expanded during the withdrawal of the effusion. I employ needles, therefore, which are only 1–2 mm. in diameter, and remove only 50 to 100 grammes per minute. By this means I have removed 3,500 grammes at one sitting, which lasted about sixty minutes, and was accompanied by no discomfort to the patient.

Dieulafoy attributes many of the disasters which follow thoracentesis to the amount which has been withdrawn, and he says we should never remove more than 1,200 grammes at one sitting, and repeat in a day or two if necessary. Believing, however, that the rapidity of the flow is more important than the amount, I think that very little danger can accompany the gentler method of operating. Of course, the siphon will not work in cases of pneumo-hydrothorax, because the flow will cease as soon as the air or gas enters the tube. The best way to fill the tube with the carbolized water is to hold it up straight with the valve open, and then gradually coil it down into a basin of the prepared fluid until it is entirely submerged. Then close the valve, and the apparatus is ready for use. In case the canula becomes plugged, the basin can be elevated above the patient, so that the siphon will work back into the chest, and thus wash out the needle. *Risel* recommends a branch tube attached to a syringe, by which any obstruction in the canula can be washed back into the chest. A still simpler method would be to wash out the canula by means of a Davidson syringe in case of stoppage. Prof. *Flint* strongly recommends the Davidson syringe as an aspirator, the suction force of the bulb being used to draw the fluid out of the chest. The great advantages of the siphon are four in number: 1. The force employed is constant and practically uniform. It can never become dangerously great. 2. The expansion of the lung and the replacement of other organs are gradual and gentle. 3. The operator has little or nothing to do after the insertion of the needle. He can sit quietly and watch the flow, and he is not tired by working a pump or hand bulb. 4. The apparatus is simple, cheap, and can be carried about in small bulk.

Examination of fluid.—Very important information may be obtained at times by an examination of the fluid withdrawn from a chest. *Boegehold* and *Quincke* report cases where an effusion, after standing some time, presented a thin, white layer on its surface, which was found to consist of strongly refracting fat-granules, also fat-drops and cells. These cases were subsequently found to be due to the malignant disease of the pleura and tuberculosis. *Foulis* describes peculiar masses of cancer-cells which are sometimes found. These cells present all stages of development, from a minute solid nucleus invested with protoplasm, to large, free, nucleated cells, and all stages of development are found in the same mass. Young cells may be seen budding from old ones. At first they appear as minute buds without a nucleus. As they increase in size, a nucleus appears, and finally they become as large as parent cells. *Dieulafoy* has found that a simple serous effusion ordinarily contains 500–600 red blood-globules per mm^3. In order for the effusion to be colored, there must be 5,000–6,000 red globules per mm^3., and blood to that amount almost always indicates future purulence of the fluid. He thinks that a purulent effusion always begins with a hemorrhagic stage, and therefore each specimen should be examined to test this point.

Empyema.—*Lister* has laid down the following rules for opening the chest in empyema, and he says that a strict observance of the same will give wonderfully good results. First, the patient's side should be washed in carbolic solution (1 to 20), and the hands and instruments of the operator as well. Then the opening into the pleura should be made under a thoroughly trustworthy spray, so that no air can enter the chest unless it be impregnated with the acid. The subsequent dressing consists of a drainage-tube about three-quarters of an inch in length, or long enough to go thoroughly into the pleural cavity. The tube may be made of rubber or metal, and should be armed with a collar of metal, to prevent its slipping in. Then apply coverings of gauze, which have been charged with a disinfectant composed of one part of carbolic acid to four parts each of resin and paraffine. It is necessary to employ pure paraffine, because the crude article acts destructively upon the caoutchouc of the mackintosh cloth which is spread over the gauze. The gauze should be laid on in eight folds or more if the drainage be excessive. The dressings may be kept in place by elastic bandages around the edge of same, and drawn tight enough to hold. Change the dressings of an adult twice daily under the spray. *Lister* claims that this treatment stops all suppuration immediately, and the after-discharge is serous.

DISEASES OF THE LUNGS.

CROUPOUS PNEUMONIA. — HYPOSTATIC PROCESSES. — EMBOLIC
PNEUMONIA.—FAT EMBOLISM.—HYPERÆMIA AND ŒDEMA.
—ATELECTASIS.—EMPHYSEMA.—GANGRENE.—NEW
GROWTHS.—PULMONARY CONSUMPTION.—
TUBERCULOSIS.

BY

FRED'K C. SHATTUCK, M.D.,

OF BOSTON,

SITING PHYSICIAN TO THE HOUSE OF THE GOOD SAMARITAN; PHYSICIAN TO OUT-PATIENTS, MASS.
GEN. HOSPITAL, AND CLINICAL INSTRUCTOR IN AUSCULTATION AND PERCUSSION
IN THE HARVARD MEDICAL SCHOOL.

CROUPOUS PNEUMONIA.

BIBLIOGRAPHY.

EBERMEISTER: Handbuch der Pathologie und Therapie des Fiebers, Leipzig,
—PEACOCK: Report on Cases of Pneumonia. St. Thomas' Hospital Reports,
V., 1875.—FISMER: Deutsches Archiv für klin. Med., Bd. XI., p. 433.—
REICH: Volkmann's Sammlung der klinischen Vorträge, No. 75.—LEICHTEN-
: The same, No. 82.—BRISTOWE: Theory and Practice of Medicine, London,
—STURGES: The Natural History and Relations of Pneumonia, London,
—WIEDERMANN: Zur Lehre von der Lungenentzündung. Deutsch. Archiv
lin. Med., Bd. XIV., p. 389.—FLINT: Pneumonic Fever; Transactions of the
State Medical Society, 1877.—COHNHEIM: Vorlesungen über allgemeine
logie, 1877.—LEYDEN: Die Ausgänge der Pneumonie. Berliner klin.
en., 1879, Nos. 20 and 21.—WELLS: Analysis of 100 Cases of Pneumonia.
nnati Lancet and Clinic, 1879, II., p. 241.—STRAUSS: Note sur un cas d'érysi-
es Bronches et du Poumon. L'Union Médical, Oct., 1879.—RITTER: Pneumo-
s. Deutsch. Archiv für klin. Med., Dec., 1879, p. 53.—HAMBURGER: Unter-
ngen über croupöse Pneumonie. Inaugural Dissert., Strassburg, 1879.

he two leading peculiarities of *Jürgensen's* article on this subject
is vigorous advocacy of the theory that pneumonia is an infectious
se, and the view that a fatal result is generally due to failure of the
In the second edition of *v. Ziemssen's* Encyclopædia, with en-
d experience and basis of facts, he emphasizes these views, as well
e various conclusions to which statistical analysis had led him in the
edition; but makes no very material changes. During the last few
, attention has been reawakened from widely different quarters to a
of cases of pneumonia characterized as "asthenic," "adynamic,"
ious," "typhoid," "pythogenic," "endemic," "epidemic," "con-
us," "erysipelatous," and the like; and the burning questions in
ion to this disease are to-day: I. Is pneumonia an infectious
se, or is it a simple inflammation? II. Is there an asthenic, etc.,
of pneumonia, distinct from the ordinary or less severe form; or is
but one form, the manifestations of which differ only according to
ntensity and concentration of the exciting cause and the power of
ance of the patient?

o the consideration of these two questions, and an attempt to indicate
ight which has been thrown on their solution since 1873, it is pro-
l largely to limit this report.

A better and more widespread understanding of the part played in
ausation of disease by bad hygiene, or, as *Simon* graphically puts it,
has naturally prepared the way for the reception of the doctrine of
nfectious nature of pneumonia; and there is no doubt that *Jürgensen's*

views on this point have gained many adherents since their promulgation. They have been strongly adopted by *Sturges* and *Flint* among others. The latter calls the disease "pneumonic fever," as does *Bernheim* (Leçons de Clin. Méd., 1877, p. 17 et seq.), and says that name is as appropriate as the term enteric applied to typhoid fever. He derives his grounds from the morbid anatomy, etiology, clinical history and treatment of the disease; and states them so clearly that, in view of the great and well-deserved respect which is universally accorded to Dr. *Flint's* opinion on medical questions, a very brief abstract of them can hardly be out of place.

MORBID ANATOMY.

It is not easy to reconcile the large quantity of the exudation; its probable derivation from the branches of the pulmonary artery; its complete removal without injury to the air vesicles; its gradual and sometimes successive extension over one or several lobes, and the partiality of the disease for the lower lobes, especially the right, with the deduction that the products in pneumonia are the result solely of a local inflammatory condition.

ETIOLOGY.

Neither local affections of the lungs, bronchial tubes, nor pleura, nor violence applied to the chest ever give rise to acute lobar pneumonia; and here it may be added that *Heidenhain* (*Virchow's Archiv*, Bd. 70, p. 441) could not succeed in exciting croupous pneumonia in dogs and rabbits artificially. The inhalation of hot and cold dry air through a canula in the trachea had absolutely no effect on the lungs or the height of a thermometer in the trachea; but moist cold and heated air caused lobular pneumonia. *Sommerbrodt* (*Virchow's Archiv*, Bd. 55), in connection with experiments on the relation of blood in the air-passages to phthisis, found that microscopically local spots of croupous pneumonia resulted from the introduction of a solution of the chloride of iron into the lungs. *Jürgensen* and *Schüppell* repeated his experiments carefully on dogs, and found that even anatomically the product differed from that of croupous pneumonia. In short, croupous pneumonia has never up to the present time been produced artificially.

"Pneumonia," Dr. *Flint* continues, "as is well-known, is not infrequently an intercurrent affection in the course of other essential fevers, namely typhus and typhoid fevers, measles, diphtheria, etc. In these instances the determining cause must be constitutional, and yet, as the affection is only an occasional complication, the determining cause involves something which does not necessarily pertain to these fevers. This something, it is reasonable to conclude, is pneumonic fever. Hence it follows that pneumonic fever may be associated with other febrile diseases. The blending of different fevers may be considered at the present time as a well-established pathological doctrine. An example with which all of us are familiar is the typho-malarial fever."

A specific cause is not as yet demonstrable, but the same remark is also true of malarial and other essential fevers. The prevalence of the disease at certain seasons of the year; the relative immunity of some climates over others; the fact that it has, at certain times and places at the South, been known to prevail to an extent entitling it to be called endemic, and the fact that at different times and places the disease has varied as regards its phenomena and rate of fatality, are all adduced as etiological arguments in favor of the specific nature of the affection. Are not the same arguments equally potent in favor of the existence of bronchitic and pharyngitic fever?

CLINICAL HISTORY.

The disease is generally ushered in by a well-marked rigor followed by fever and a rapid and pronounced rise in the temperature, which fever cannot be symptomatic merely, for the reason that often no physical signs can be obtained until several days have elapsed; that the pulse and temperature afford no indication of the extent or extension of the disease; and that defervescence often takes place decidedly before it is possible to detect any change in the physical signs—in other words, that defervescence is not determined by conditions which relate to the local lesion. In fact it is a self-limited disease. The analogy to typhoid fever, which in several points of view is apparent, is further shown by the frequent occurrence in pneumonic fever of what are known as typhoid symptoms. It is true that they occur in various diseases, but they are far more frequent in pneumonic fever than in any other disease, except typhoid and typhus fevers. They cannot be attributed to the interruption of the respiratory function, for they are rare in such diseases as pleurisy, capillary bronchitis, and asthma; but are undoubtedly due to the fever, irrespective of the pulmonary affection.

Pneumonic fever, again, has no immediate tendency to relapse; in a large number of cases recorded by Dr. *Flint* relapse is not noted in a single instance, nor does he recall such an occurrence in any unrecorded case. It never persists in a chronic form; clinical experience shows that it does not eventuate in phthisis, and if death does not take place from the disease, its complications, or its accidents, recovery follows without any risk of the persistence of the local affection in a chronic form.

TREATMENT.

The influence over the disease of antipyretic remedies—quinine, salicylic acid compounds, and cold water—speaks also in favor of the doctrine of essential fever.

Dr. *Flint* has seen the disease rendered abortive in a certain proportion of cases by 20–40 grains of quinine daily, and when this result has not followed, the disease is often favorably modified in a greater degree than by smaller doses.

Finally he defines the affection as "a fever characterized anatomically by an abundant exudative deposit in the air-vesicles of a single lobe, or of two, and sometimes three lobes of the lungs with, in general, circumscribed bronchitis and dry pleurisy. It is a fever which rapidly reaches its maximum of intensity, and has a short course, the duration averaging about eleven days. It proves fatal chiefly in consequence of associated diseases, complications, or accidents, and the mode of dying is by asthenia. It is non-communicable, and depends on a cause, or causes, specific in character, the nature of which is at present unknown, but having relations to season and climate. It sometimes aborts spontaneously; and it is in some instances arrested by remedies. If not arrested it may be favorably modified, its duration abridged, and the danger to life diminished by treatment addressed, not to the pulmonary affection, but to the fever."

It will be observed that *Flint's* arguments are essentially the same as those of *Jürgensen,* though they are more elaborate as well as more concise. In the presentation of the other side free use is made of critiques on *Jürgensen* and *Sturges* which appeared in the *Brit. and For. Med.-Chir. Review* for January, 1877, and on *Sturges* in the *Edin. Med. Jour.* for the same month and year.

Both authors—and presumably *Flint*—follow *Hirsch* and *v. Ziemssen,* of Berlin, who propounded his views in 1858, based largely on the London mortality returns of sixteen years (1840–56). *Buchan* and *Arthur Mitchell* (*Journal of the Scottish Meteorological Society,* July 1874–75, p. 190) have investigated the same subject on the basis of thirty years of the same returns and have not found *v. Ziemssen's* views in any way supported. On the contrary, they have found the mortality curve for pneumonia to be very steadily marked from year to year, and to be essentially the same as that from bronchitis. The pneumonia curve also differs *in toto* from those of typhus and typhoid, or even simple continued fever, as well as from that of pleurisy, the curve of the latter being somewhat analogous to that of rheumatism and pericarditis. There is, therefore, nothing in these statistics—the most perfect in the world and extending over a longer period than any others—that lends the slightest support to the theory that pneumonia is a specific fever of any kind, or indeed anything else than an inflammation pure and simple. Prior to 1869 all fevers were classed together in the London mortality returns, and between 1840 and 1856 typhus fever prevailed in that city more or less. We know that typhus and typhoid have a very different seasonal mortality, that both of them differ very materially from that of pneumonia, and, therefore, how much value to attach to the curve figured on page 29 of the translation of volume V.

It is true that in pneumonia the fever often abruptly ceases before the local lesion is resolved; in a healthy subject this is by no means the rule, but, be that as it may, the rapid cessation of a frequently very high temperature with the continuance of the local lesion often for a very long time is a daily occurrence in embolic pneumonia, which is purely a local process and could not possibly be supposed to have even the most remote connection with any form of specific fever; and, indeed, it requires but a slight acquaintance with pyrexial processes to discover that the period of convalescence or repair is never pyrexial, or, as *Burdon-Sanderson* has stated, "does not form part of the febrile process."

As for *Flint's* assertions that pneumonia never persists in a chronic form, and that it never leads to phthisis, it can only be said that many other first-rate authorities do not agree with him. One of the causes of interstitial pneumonia is claimed to be a croupous exudation which did not undergo absorption. *Jürgensen* himself says that this sequence, though not common, cannot be doubted by a physician of large experience (2d Ed. *v. Ziemssen's* Encyclopædia).

As was hinted above, many of the arguments for the specific nature of pneumonia have a very wide application, and speak quite as strongly for the specific nature of bronchitis, pharyngitis, and some other affections. Very likely they do partake of this nature at times, but there can be no question that they are very often simple inflammations. So with pneumonia; may it not be that the disease is sometimes an essential fever, sometimes a simple inflammation? Further than this I do not see how we can go at present. We have had a pathology of the humors and of the solids; we now bid fair to have a pathology of the specifics.

Klebs (*Arch. f. Exp. Path.*, Bd. IV., p. 420) figures and describes an organism, *monas pulmonale*, which he found in the ventricular fluid of patients who died of pneumonia, and in the sputa during life, and by injecting which in rabbits he has produced lobular pneumonia, with which was associated meningitis, nephritis, etc. He thinks that this explains the frequent association of pneumonia and erysipelas with the above affections, as well as pleuritis and pericarditis. These observations are as yet entirely lacking in confirmation by others, and caution is never more imperative than in connection with these low forms of life. That pneumonia may apparently be set up by sewer gas is shown by a striking outbreak (*Med. Times and Gaz.*, April 4th and June 20th, 1874), quoted nearly as abstracted in the *Irish Hospital Gazette* for Nov. 1st, 1874.

Sewer-Gas Pneumonia.—On Saturday, March 14th, the parish sewer in the road nearly opposite a first-class boys' school, at East Sheen, near London, was opened by order of the sanitary authorities for the purpose of inserting a ventilator protected by a charcoal screen. The headmaster remonstrated, and backed up his own objections by a certificate signed by several eminent medical men, including Sir *William Jenner*, two of whose sons were students at the school. Sir *William Jenner* especially mentioned the danger of pneumonia in connection with the probable escape of sewer-gas in the vicinity of the school. The authorities persisted in their scheme with a trifling modification. March 20th, a high tide in the Thames blocked up the mouth of the sewer, and the compressed gases forced an opening through the ventilator. The rooms of the school facing the road were filled with a foul-smelling sewer air. Next morning, a boy sleeping in one of these rooms was taken seriously ill with pneumonia; on the evening of the same day two other boys and two servants became similarly affected. One of the servants ultimately died. The school was broken up, and the authorities were ordered to remove the ventilator and close the opening on the evening of the 21st, after which all smell ceased, and no additional cases of illness of any kind occurred in the house. For fifteen years previously, no illness attributable to bad drainage had occurred, and competent inspectors pronounced the sanitary arrangements of the house excellent.

The fact that pneumonia was predicted lends additional interest to the above report. We are not given to understand that the cases presented any peculiar clinical features.

II. What is the position of the severe form of pneumonia called typhoid, asthenic, adynamic, nervous—does it differ etiologically from the common form or not? *Jürgensen* puts this question (2d Ed. v. *Ziemssen's* Encyclopædia), discusses it briefly, and answers: "Personally I do not hesitate to express my firm conviction that there is but one pneumonic poison; it is just as with roses: either each kind of rose forms a species, or there is but one species including many varieties."

Laennec (De l'auscultation médiate, 4th Ed., Vol. I., p. 595) observed and described an affection which prevailed among the conscripts of 1814, and was called by him epidemic pneumonia. A remarkable epidemic of contagious pleuro-pneumonia started in Canada during the war of 1812, and spread through the United States far into the South, though all facts of this kind seem to have been lost sight of under the universal prevalence of the doctrine of the simple inflammatory nature of the disease, until *Hirsch* formed a chronological list of similar epidemics occurring between 1521 and 1858.

Within a few years the subject has been brought prominently forward by *Liebermeister*, *Friedreich*, *Fismer*, *Leichtenstern*, and others; and our knowledge has been enriched by the report of carefully observed and most interesting local outbreaks which are worthy of being dwelt upon at some length.

In the *American Journal of the Medical Sciences* for January, 1876, *Rodman* describes a series of cases observed by him in the prison at Frankfort, Ky., at a time when there was but little pneumonia about, and it was only among the prisoners that this form of the disease occurred. Between January 1st and February 24th, 1875, 16 cases of ordinary pneumonia occurred in the prison, one of which was fatal. About the last week in February, it was evident that the type of the disease had radically changed, and the number of cases was greatly increased. The whole number of cases of pneumonia from January 1st to July 1st was 118; of these, 98 were of a peculiar type, and 25 of the 98 were fatal. The disease is said to have resembled closely ordinary pneumonia, both as to physical signs and post-mortem appearances, though it is mentioned that the sputa were generally either pure blood or a dirty brownish-black in color.

Pain was not a prominent symptom, and in many cases the lung-consolidation was revealed only by physical exploration. The pleura was implicated oftener than in simple pneumonia, and in several cases circumscribed empyema was found. The bowels did not seem to be much affected, but the dejecta were excessively offensive. The liver was enlarged, and, in fatal cases, filled with dark syrupy blood; no mention is made of the spleen or kidneys. Men died with hepatization of but a single lobe, and the disease was very treacherous as regards prognosis. The average temperature seldom ranged as high as in ordinary pneumonia. The patients might seem better as regards the pulse, respiration, temperature, cough, and expectoration; but take a sudden change for the worse and die. Carbonate of ammonia, on which previous experience has led Dr. *Rodman* to rely greatly, proved useless; and much better results seemed to follow the administration of quinine, and the tincture of the chloride of iron, with liberal diet and free stimulation. Convalescence was very protracted.

The cell-house is 310 feet long, 43 feet wide, and 75 feet high, and contains 648 cells, each of which contains 170⅔ cubic feet of air; and the cells, like the cell-house, are very poorly ventilated. Feb. 1st, 694 men were confined in these cells, and during this month the number of prisoners received was so much in excess of those discharged, that by March 1st 735 men occupied these same cells. There are six tiers of cells on each side of the house, but those next the roof, 108 cells, cannot be used, it being almost certain death for any one to sleep in them, especially in summer. It was consequently necessary to double the men in some of the cells, and this was done in the uppermost tiers. The strongest cells are near the floor, and are reserved for the whites, who are more apt to escape than the negroes. Every man takes a night bucket with him to his cell, only intended to be used in case of emergency; but rather than wait their turn at the privy, 400 of the men will use their buckets in the cells between dark and daylight in spite of severe penalties. The basement tier was comparatively free from bad odors, and there was less sickness there than in any other part of the house. The odor increased with each tier, and, when the top was reached, the stench was almost unbearable by one unaccustomed to it. Of the 25 who died, 24 were negroes (occupying the upper cells) and 14 were new prisoners, reckoning those as new who were received after January 1st. The new prisoners were to the old numerically as one to four. These facts show the causative agency of the bad air, and illustrate the greater susceptibility of a stranger over a native, so to speak, to zymotic poison. We are all familiar with the frightful ravages caused by the importation of measles into the Fiji Islands not many years since. We shall have further evidence to bring on this point later.

Overcrowding and consequent intensification of the poison seems to have been the exciting cause of this outbreak, for the condition of the men was in other respects precisely the same as during the time that the pneumonia presented no distinctions from the ordinary type. *Rodman* states that twice before in the history of the prison pneumonia prevailed to a fearfully fatal extent, and in both instances coincided with marked increase in the number of the prisoners.

Dr. *L. Dahl* (*Dublin Journal*, I., 1875, p. 405) describes an outbreak of pneumonia in the Akerhus prison, Christiania, which commenced in Dec., 1866, and terminated in May, 1867. Among an average of 360 prisoners in that period, 62 cases occurred with 15 deaths. The weather was very cold at the height of the epidemic, but the prisoners who worked in-doors were about equally attacked with those who worked in the open air.* Prof. *Boeck* attributed the outbreak chiefly to *overcrowding*, and Dr. *Dahl* calls attention to the improved state of affairs coincident with a diminution in the number of the prisoners from 387, Jan. 1st, to 278, Dec. 31st, 1867. A former similar outbreak occurred in the prison in 1847, and coincided with a prevalence of scurvy.

Kühn (*Deutsch. Arch. f. klin. Med.*, 1878, p. 348, and *Berliner klin. Wochenschrift*, Sept. 15th, 1879) contributes reports of outbreaks in the House of Correction at Moringen, Hanover, in the years 1875 to 1878, which differ in some respects from that of *Rodman*. In the year 1874, it was noticed that there was an unusual amount of sickness among the prisoners, and that this increase was chiefly in diseases of the respiratory organs. Pneumonia ran a peculiar course, much more like that of an infectious disease than of the ordinary type. There were also a considerable number of cases of an ill-defined febrile affection, the onset of which was preceded by pain in the head and back, and malaise, which ran a mild course with sudden defervescence about the tenth day, and was followed by tedious convalescence. The most prominent symptoms were either those of gastric catarrh or of bronchitis and lobular pneumonia. Enlargement of the spleen was nearly constant. In 1875, cases of illness were still more numerous, the maximum being reached in February, when ten and a half per cent was the daily proportion of the sick. During the year there were only eleven cases of pneumonia of the ordinary type, but during the first nine months there were eighty cases of general febrile disturbance, more severe and, at the same time, less vague in character than those of the year before. After prodromata lasting from five to eight days, rigors and a rapid rise of the temperature to 103–104.5° came on; during the next few days simple sore throat, enlargement of the spleen, and, in the severer cases, albumen in the urine appeared; but the belly presented neither spots, distention, nor tenderness. Not till the third or fourth day could consolidation of the lung be detected; this consolidation was generally lobar, very often of the upper or middle lobe, and sometimes disappeared without ever becoming complete, to reappear in another portion of the lung, thus resembling the so-called "wandering or migratory pneumonia." Foci of lobular pneumonia were very common in the upper lobes, both with and without a lobar process in the lower lobes. Severe pleuritic symptoms were constant; meningitis was noted in five cases, and the cerebral symptoms in general were second in prominence only to the pulmonary. Pericarditis occurred in twenty-five per cent of all the cases, and the albuminuria generally continued as long as the pyrexia. It was present in every case in which the temperature surpassed 104°. In two-thirds of the cases which proved fatal from the direct effects of the disease, the solitary follicles and Peyer's patches were swollen, but not ulcerated. In fact, the symptoms were those of severe general blood poisoning, but the course was decidedly shorter than that of typhoid fever, and the temperature curves were more like that of pneumonia. Sixteen cases were fatal, and in fourteen of these the pneumonia was exclusively of the lobar variety. That the affection was contagious to a degree is shown by its appearance in nurses and chronic patients in the hospital; and above all, by the fact that one of the overseers took it home to his family who lived at some distance and never came into direct communication with the prison.

Kühn himself was laid up several days with malaise, and about the same time his coachman, whose duty it was to brush his clothes, fell ill. Then the housemaid who undertook this duty was taken sick, and finally his daughter, a child of four years; the two latter cases being quite severe, and followed by tedious convalescence and loss of the hair. As soon as the housemaid was sufficiently well she went to her friends, who lived some miles distant, taking with her articles of clothing which had been used and kept in her room during her illness. She occupied the same bed with her sister, who, just eight days after the arrival of the sister at home, was also taken ill in precisely the same way.

The above facts taken together lead *Kühn* to formulate the following conclusion: "*There is then a contagious affection, the course of which is the same as that of primary asthenic pneumonia, but which is entirely distinct from ordinary pneumonia, and differs no less from it than does typhoid from typhus fever.*"

In this epidemic again we find that overcrowding seems to be the exciting cause. The prisoners were chiefly incorrigible tramps and beggars, and averaged about one hundred and eighty in number previous to 1874. In that year the influence of hard times began to be felt and the number of prisoners was about one-third larger than could be properly accommodated. New buildings were undertaken, and by the end of 1876 there was room for six hundred prisoners, but commitments increased in still larger proportion, and overcrowding was constant, continuing at least into 1878, though in a less degree. It is stated that the sanitary arrangements were excellent, but on these points the account is not as full and clear as could be desired, and we are told that wooden buckets were used in many of the dormitories for the reception of the renal and alvine discharges. Great pains seem to have been taken to keep these buckets clean, and it is certain that the hygiene of the place was vastly superior to that of the Frankfort Prison. It will also be noted that the German mortality was less. In the town of Moringen itself, there was during the year 1875 decidedly less typhoid fever than in previous years; but diseases of the respiratory organs were rife, as well among adults as children, and partook in their course and complications somewhat of the nature of the prison disease.

During 1876, 1877, and 1878, this form of pneumonia continued to be the prevailing disease in the institution. According to *Jürgensen*, pneumonia constitutes 3% of all diseases, and 6.4% of all diseases of the internal organs; but in 1878 the proportions in the Moringen workhouse were 8.81% and 17% respectively; catarrhal, cheesy, and secondary pneumonia being carefully excluded from the statistics. The number of cases was fifty-eight, seventy per cent of which occurred during the two months of March and April. A few of these cases differed in no respect from ordinary pneumonia, but the great majority were of the previously described type. Eight were fatal, a proportion of 13.80%. The post-mortem appearances were much the same as in 1875, except that the swelling of the intestinal glands was decidedly less marked. The less intensity of the epidemic of 1878 is also shown by the fact that though a nurse in charge of a ward containing several pneumonia patients was taken down with the disease, there is no evidence that it was communicated to any person outside of the institution. In 1877, 71.82%, and in 1878, 77.58% of the cases were in those who had been less than six months inmates of the prison, while only 31.99% of the whole number of prisoners had been there six months or more.

Grimshaw and *Moore* (*Dublin Journal*, I., 1875, p. 399), call attention to a type of pneumonia which was common in Dublin during the warm months of 1874, and which they characterize as "pythogenic." The distinctive symptoms were sudden invasion, the frequency with which the disease was arrested in the early stage, and a less liability *constantly* to attack the lower lobe of the right lung. It is stated that the disease is not usually very fatal, but that an accurate percentage of mortality cannot be given because of the difficulty in distinguishing some of the cases from enteric fever on the one hand, and true pneumonia on the other. All of these patients came from houses the sanitary condition of which was far from satisfactory. They refer to an epidemic of pleuro-pneumonia in some ships of the British Mediterranean fleet in 1860; the disease was of an asthenic type accompanied with great congestion of the lungs, and, in many cases in the ship chiefly affected, with scorbutic symptoms. Overcrowding and defective ventilation were the most tangible causes, and there were good grounds for supposing that the affection was communicated by the sick landed from the vessels to the patients in the Maltese hospitals.

Some of the conclusions drawn by the authors in their paper are as follows:

1. That the bibliography of pneumonia indicates the existence of a form of the disease which arises under miasmatic influences and is contagious.
2. That its etiology justifies us in regarding it as a zymotic affection and in terming it "pythogenic" pneumonia.
3. That pythogenic pneumonia presents peculiar clinical features which enable us to distinguish it from ordinary pneumonia.
4. That whereas ordinary pneumonia is specially prevalent during a

continuance of cold, dry weather with high winds, and extreme variations in temperature, pythogenic pneumonia reaches its maximum during tolerably *warm* weather, accompanied with a dry air, deficient rainfall, hot sun, and rapid evaporation.

Barella (*Bull. de l'Acad. de Méd. de Belg.*, No. 2, 1877) supports the views of *Grimshaw* and *Moore*, and adds that the disease has often at the beginning abdominal symptoms which make it look much like typhoid fever. In this connection it may be well to refer to ***Murchison's*** teaching, that in cases where the diagnosis is doubtful between pneumonia and typhoid fever, the appearance of consolidation before the tenth day of the disease points to the former affection.

Hardwiche (*Gaz. Méd. de Paris*, 1876, p. 515) reports some cases in which the disease seemed to be contagious, but they are so lacking in details as to be of little value.

Kelemen (*Pester Med. Chi. Presse*, XII., 1776, 45–46) alludes to four cases of "migratory pneumonia," and adds one of his own. *Waldenburg* compares the disease to erysipelas migrans, and *Friedreich* believes that they are practically the same thing, as he has seen many cases of migratory pneumonia during the prevalence of erysipelas. In ordinary pneumonia one, two, or (very rarely) three foci are co-existent and disappear together, even if they do not begin at the same time; but in the migratory variety one focus makes its appearance and is followed by another, and another, the new one not appearing until its predecessor has vanished. The foci may run through all three stages, or never go beyond the stage of engorgement. In these five cases the maximum number of foci was ten, the minimum four.

The limits and purposes of this report do not permit of anything more than a passing allusion to the typhoid pneumonia of which so much was heard during the late civil war, and to the possibility of some connection between the pleuro-pneumonia of cattle and certain epidemics in man.

At what conclusions as to the etiology and pathology of pneumonia do the above facts, in connection with other knowledge and experience, enable us to arrive?

A good deal can be said in favor of each of the four following statements, and the profession doubtless comprises adherents of each:

1. True pneumonia is simply a local inflammatory disease, but many general constitutional affections are at times accompanied by a pulmonary lesion which bears a close resemblance to simple pneumonia, though it is in reality a part of the constitutional process; the results of this process happening, for some cause unknown, to be localized largely in the lungs.

2. Pneumonia is often simply an inflammatory disease, but may depend on various miasmatic influences and then present decided variations in its course and symptoms.

3. Pneumonia is never a simple inflammation, but always an infectious disease arising from a single specific cause as yet unknown, and

varying in its effects according to the intensity of the poison, the power of resistance and the surroundings of the patient.

4. Pneumonia is always an infectious disease arising from dual, possibly plural, specific causes. Of these causes one produces sthenic fever, of more or less definite type, and with localized lesions, while the other or others produce an asthenic fever far less definite in type and with more or less extensive lesions of other important organs and parts as well as of the lungs.

Of these statements the second is that which to me individually seems most in accordance with the facts at our disposal to-day, though any statement as to a matter of this nature is merely a halting-place, as it were, on our journey, from which we cast our eyes over that part which lies behind, and mentally remeasure and consider the length and obstacles of the path before us.

Other aspects of the disease require but brief mention. *Marshall* (*Medical Record*, N. Y., Vol. 17, 1880, p. 185) reports a case of pneumonia with recovery after a temperature of 110°. It is interesting to note that, while *Jürgensen* and *Sturges* agree in the main as to the infectious nature of pneumonia, they differ as to treatment. The latter advocates moderate bleeding as a means of relief to the dyspnœa, even after a considerable extent of the lung has become solid—so late as the third or fourth day, but says that the cold bath is less applicable to pneumonia than to other febrile conditions where the functional defect is less ominous, because he regards exceptionally high temperature in pneumonia as simply an indication of a necessarily fatal condition of the lung, and sees no reason to employ cold bathing as long as the pyrexia is moderate. As to the utility of stimulants to tide over a period of depression, he agrees with *Jürgensen*.

Hamburger reports that in the winter of 1877–78 seventy-seven cases of croupous pneumonia were treated at the Strassburg hospital. Of these, seven died, and the heart was healthy in every case—in other words, death was not due to cardiac failure.

HYPOSTATIC PROCESSES IN THE LUNGS.

BIBLIOGRAPHY.

Christensen, Ludwig: Einiges über Lungenentzündungen beim acuten Gelenkrheumatismus, 1868.—**Ramm, Heinrich**: Ueber die Retraction der Lungenränder, 1869.—**Paulsen, Eduard**: Ueber die Verschiedenheiten des räumlichen Inhalts des Thorax im Stehen und Liegen, 1874.—**Erhardt, Wilhelm**: Ueber den nachtheiligen Einfluss einer anhaltenden Rückenlage auf die Mechanik der Athmung und deren Bedeutung für die Enstehung des Lungencollapses, 1875.

(The following is translated from the second edition *v. Ziemssen's* Encyclopædia.)

In *diagnosis* there are two great sources of error.

A *croupous pneumonia,* which supervenes on a long-standing malady attended with considerable fever, may give rise to such slight symptoms as to escape notice, unless examinations are often and carefully made. The same remark is true of croupous pneumonia in feeble and aged persons, especially when emphysema and chronic bronchitis are also present. If, under these circumstances, *bilateral* consolidation is found over the lower lobes, the chances are very strong that the condition is one of hypostasis. *We must not, however, entirely lose sight of the facts that unilateral hypostasis and bilateral croupous pneumonia are sometimes met with.* A diagnosis which is based merely on the ground that one only, or that both sides are involved, is not much better than a guess. In many, yes, in most cases, the history or other points which one will have noted if the cases have been some time under observation, will throw all necessary light upon them.

It is well known that *collapse* in a greater or less degree always occurs at the anterior borders, and the lower and posterior portions of the lungs of patients with feeble respiration, who are compelled to lie on their backs for long periods. A good deal of attention has been devoted to this point now for a series of years at the *Kiel* clinic, and *Bartels* has inspired a number of dissertations on the subject containing the results of his experience (see bibliography). Collapse of the lower and posterior portions of the lungs occurs in connection with various apparently quite distinct diseases, provided that they have the common factors of superficial respiration and the constant maintenance of the same bodily position; and this collapse may be sufficiently marked to give rise to physical signs if the disease be of long duration. *Portions of pulmonary tissue thus collapsed may become the seat of hypostasis and catarrhal pneumonia,* the latter either in isolated patches or involving the whole area. Both conditions may be associated, or either may exist independently of the other.

Bartels has shown that when the body is horizontal, the muscles which serve to dilate the thorax work at a disadvantage as compared with either the erect or the sitting posture. In the prone position the contents of the abdominal cavity gravitate upwards and press against the diaphragm; in standing or sitting, on the other hand, the exact reverse is the case, and all the abdominal viscera gravitate downwards toward the pelvis. The anterior wall of the belly serves as a support, but when the body is horizontal on the back, the weight comes entirely on the spinal column, the lateral surface of the abdomen, and the diaphragm. Of all these surfaces the diaphragm is that which offers least resistance to the pressure, is consequently the first to give way to it, and thus encroaches on the pulmonary space. In the prone position again, the distance between the crest of the ilium and the lower ribs is increased, and the diaphragm is put at a greater disadvantage by this increased tension of the abdominal wall. The observation that the vital capacity of the lungs is less in the recumbent than in the erect position (*Hutchinson, Wintrich, Panum, Pfahl-Quincke*) is entirely in accordance with these facts. In very muscular persons, the difference is not indeed very great, certainly during the short time required to respire a few times while an observation is taken with the spirometer, but in rather feeble persons it is as great as 400–600 c. cm., even when the full inspiratory force has been called into play only a few times and no real fatigue has been incurred.

A plain statement of the matter may be made as follows: A change from the erect to the recumbent position increases the forces which oppose respiration to such a degree that a decidedly greater demand is made upon the muscular power to counteract these forces in a measure, even for a brief period; to counteract these entirely is impossible. If the dilatation of the thorax incident to inspiration is lessened, or, in other words, if the difference in the atmospheric pressure within and without the thorax is diminished, the lungs tend to approximate to the position at which their elasticity is exhausted, and this the more in proportion as the respiration is feeble. The conditions which experience has taught us favor collapse are pain on inspiration from peritonitis, double pleurisy, or acute rheumatism, feebleness of the respiration from long-continued fever, and loss of muscular power from tonic spasm, such as that of tetanus. If something be superadded to these conditions which favors nutritive changes in the walls of the vessels—hypostasis, the escape of the constituents of the blood, both formed and amorphous—is the result. That this whole process may go through its several stages in a short period of time is proved by the existence of well-marked collapse and hypostasis in a patient who died from tetanus on the fourth day after seizure (*Erhardt*). When favorable conditions for the development of cartarrhal pneumonia are present, this process is also set up, either in isolated patches, or uniformly distributed over the whole collapsed portion of lung.

Theoretically, doubtless, collapse, hypostasis, and catarrhal pneumonia are quite distinct processes, but their underlying causes are so often associated during life and their physical signs are so similar that it is often practically impossible to differentiate them at the bedside.

EMBOLIC PNEUMONIA.

BIBLIOGRAPHY.

COHNHEIM and LITTEN: Ueber die Folgen der Embolie der Lungenarterie. Virchow's Archiv, Bd. 65.—PENZOLDT: Ueber den hämorrhagischen Infarkt der Lunge bei Herzkranken. Deutsch. Archiv für klin. Med., Bd. XII., 1874.—GERHARDT: Der hämorrhagische Infarkt. Volkmann's Sammlung der klinischen Vorträge, Nr. 91.—KOSSUCHIN: Zur Lehre von dem embolischen Infarkte. Virchow's Archiv, Bd. 67, p. 449.—KÜTTNER: Beitrag zur Kenntniss der Kreislaufsverhältnisse der Säugethierlunge. Virchow's Archiv, Bd. 73, p. 476.—LICHTHEIM:

Versuche über Lungenatelektase. Archiv für experimentelle Pathologie, Bd. X., 1878.—LITTEN: Untersuchungen über den hämorrhagischen Infarkt and über die Wirkung arterieller Anämie auf das lebende Gewebe. Deutsch. Zeitschrift der klin. Med., No. 2, p. 131, 1879.

PATHOGENESIS AND ETIOLOGY.

As to the question whether hæmorrhagic infarction is invariably due to embolism, both *Jürgensen* and *Gerhardt* call attention to the fact that sometimes no embolus is to be found. That state of things is more common during the course of some infectious diseases, and thus naturally suggests the possibility that nutritive disturbance has so altered the walls of the vessels within a limited area of the lung as to allow the escape, not only of the coagulable plasma, but also of the red blood-corpuscles. This explanation must, of course, remain purely hypothetical until the existence of such nutritive disturbances is proved.

Our authors are also agreed as to the statement that those valvular lesions are most apt to lead to thrombosis in the systemic veins which bring about accumulation of blood in the same: similar results are liable to follow degeneration of the cardiac walls, especially when, as *Jürgensen* has found to be the case in Tübingen, such degeneration is predominant in the right heart. The size of an embolus does not alone determine the extent of the resulting infarction, for the greater or less supply of blood from other sources to the occluded vascular territory must also be taken into account; and in the case of metastatic abscess, there is still another factor to be considered: septic action, namely, may extend by contiguity into tissue supplied by arteries, other than that which is occluded.

As to the *method of development* of hæmorrrhagic infarction, our knowledge has been materially increased these last years. *Jürgensen* states that he regards the views of *Cohnheim* as unassailable, and these views assigned a very important part in the production of infarction to accumulation and stasis of blood in the pulmonary veins, due, simply, to complete loss of arterial pressure in the territory of the occluded artery. *Litten's* latest experiments (1879) agree entirely with those of *Küttner* and *Lichtheim*, and essentially modify the results of his and *Cohnheim's* experiments expounded in the second German edition of *v. Ziemssen's* Cyclopædia. He finds, namely, that in rabbits and dogs, though the hilus of one lung be ligatured *en masse*, infarction takes place, and a certain circulation is carried on through the arteries which supply the pleura, mediastinum, and pericardium. Venous accumulation cannot, therefore, be the cause of infarction, inasmuch as it may occur when the pulmonary veins are tied. *Küttner* also showed that there is pretty free communication in the capillaries between the branches of the pulmonary and bronchial arteries. He tied the pulmonary artery and veins of one lung, and then introduced considerable quantities of fluid, with cinnabar in suspension, gradually into the circulation. The pigment was found in the branches and trunk of the ligated artery, in the capillaries of the alveolar walls, and in the pulmonary veins, and the bronchial arteries were dilated. The whole tract of the pulmonary artery and veins were filled with plugs containing the pigment. *Cohnheim* and *Litten* used an aqueous solution of aniline-blue in their experiments of 1875, a pigment which we now know is thrown down in the smaller arteries and veins, and plugs them. They were thus led to the erroneous conclusion that the branches of the pulmonary artery are practically terminal arteries, and have no communication with the bronchial artery.

As proof that backward circulation does not take place in the pulmonary veins, *Litten* adduces the fact that when the pulmonary and bronchial arteries, as well as those external arteries which communicate with the lung, are all tied, that is when *all* arterial supply is cut off from the lung, while the pulmonary veins are left open, engorgement and infarction do *not* take place. But if the pulmonary artery alone, or the pulmonary and bronchial, or the whole hilus be tied, these results are constant. Physiologically, *i. e.*, with free circulation, the blood-pressure in the pulmonary artery is all sufficient to overcome the resistance to the onward flow in the capillaries, which, in the lungs, are unusually large, and offer less resistance than capillaries in general. But when the pressure in the pulmonary artery ceases from occlusion of that vessel, the collateral circulation through the bronchial and external arteries is sufficient, indeed, to prevent a backward current in the veins, though not sufficient to force the blood onward to the right ventricle rapidly enough to prevent hyperæmia and diapedesis. The capillaries and smaller veins are in precisely the same condition of distension and dilatation as when the vein into which they empty is narrowed in calibre. *Litten* found the same results in experiments on the kidney and spleen. *Infarction does not take place, in short, unless arterial blood from some source is still sent into the part after its main supply is cut off.*

In illustration of the great powers of resistance of the pulmonary artery to disturbances of nutrition, and of the different results of sudden and gradual occlusion of that vessel, *Jürgensen* reports the following case:

A woman of 54 died with general dropsy dependent on degeneration of the walls of the heart. On autopsy, the main trunk of the right pulmonary artery and its larger branches were found completely filled with a coagulum as thick as the finger, partially adherent to the wall of the vessel, laminated, externally grayish, internally reddish-black in color. The main coagulum was about 4 cm. long, and sent off numerous fresh offshoots into the lateral branches of the vessel. The intima was very atheromatous, thickened, and fatty in spots. The right lung was simply anæmic and œdematous, and entirely free from infarction. The condition of the thrombus clearly indicated that occlusion must have been gradual. The accumulation of blood in the systemic veins was so great that, when the large trunks at the root of the neck were incised, the blood spurted out in a stream three inches in height. Toward the last the circulation in the lung must have been excessively slight, and yet no marked tissue lesions were found.

The writer of this article has reported a somewhat similar case (*Boston Medical and Surgical Journal*, 1880, I., p. 242). The patient, becoming insane, was sent to an asylum, and was there so uncontrollable that it was necessary to put him into a padded room; and yet there was at the time almost complete occlusion of the right pulmonary artery, beside chronic disease of both lungs.

SYMPTOMATOLOGY AND DIAGNOSIS.

In the above case there was an hereditary tendency to insanity; but it is interesting to note that *Gerhardt* seems frequently to have seen cerebral symptoms supervene on embolism of the pulmonary artery in patients with weak hearts. According to his experience, loss of consciousness is very apt to be the first symptom of extensive embolism, whether death occurs soon after, or not. He finds that there is great variation in the degree of the cerebral symptoms: there may be a fainting fit, or merely transitory disturbance of some of the special senses. In one case the sense of hearing remained intact, while that of sight was lost. More or less well-marked signs of cardiac weakness follow the lodgment of the embolus, and it is said that an increase in the area of cardiac dulness is demonstrable. The brain symptoms are attributed to cerebral arterial anæmia.

If the embolus be very large, death may be almost instantaneous, or may be preceded by collapse, intense dyspnœa, or even convulsions. Such deaths are not very uncommon after confinement, and have been reported in increased numbers of late years. Though it is the rule that the temperature does not generally rise for several days after the occurrence of embolism, *Jürgensen* states it cannot be denied that fever attributable to this cause sometimes appears very soon. He agrees with *Gerhardt* that the character of the sputum is not absolutely pathognomonic of infarction. If the expectoration is moderately copious, careful examination will always enable us to distinguish it from that of croupous pneumonia; but that we may be deceived in other ways is shown by the following case:

A woman of 34 came under treatment with right hemiplegia, loss of the power of articulation, and aphasia. She was first seen ten days before her death, and the leading points of the clinical diagnosis were confirmed at the autopsy: failure of the heart from mitral stenosis and insufficiency, and great dilatation of the right cavities. A pleuritic effusion reaching as high as the third rib on the right side was also detected, and part of the expectoration was precisely like that due to hæmorrhagic infarction. This, and the presence of all the conditions of thrombosis led to the diagnosis of infarction in the right lung. The day before death a bloody sputum was thrown out, of the same character as before. Neither thrombus nor embolus was found in the lungs, but the bronchi contained a dark-colored secretion mixed with blood, and there was hæmorrhagic pleurisy. In the *left* auricle was a red, partially softened thrombus.

According to *Gerhardt*, the character of the sputum in cancer and echinococcus of the lung may also be similar to that after infarction. After the lapse of two or three weeks, hæmatoidin, both in the form of granules and crystals, takes the place of the blood-corpuscles. Bloody expectoration may be entirely lacking in hæmorrhagic infarction: its absence was noted by *Gerhardt* in two cases out of fifteen.

The authorities are not unanimous as to the size which must be attained by an infarction before it will give rise to physical signs. *Gerhardt* expects to find decided dulness on percussion as a rule over an infarction which is seated at the periphery of the lung, and involves an area of from one to four cubic inches. *Jürgensen* does not doubt that this may be the case with so experienced and conscientious an observer as *Gerhardt*, but questions whether others will succeed so well, even when the dull area is increased by a zone of œdematous tissue around the infarction. If emphysema is present to any marked degree, it may be difficult to determine positively the existence of even pretty extensive consolidation. *Laennec*, *Skoda*, and *Hopf* are all very guarded in their statements on this point.

TREATMENT.

Prophylaxis deserves passing mention in cases of peripheral thrombosis. Patients should be kept as quiet as possible until the thrombus has disappeared, for bodily movements tend to detach it and allow its being swept into the circulation. *Gerhardt* recommends large doses of morphia subcutaneously to relieve the attacks of dyspnœa, and musk and alcoholic stimulants may be used to excite the heart.

FAT EMBOLISM OF THE LUNGS.

BIBLIOGRAPHY.

ZENKER: Beiträge zur norm. und path. Anat. der Lunge. Dresden, 1862.—E. WAGNER: Die Capillarembolie mit flüssigem Fett. Arch. f. Heilkunde, III., p. 241, 1862. The same, IV., p. 146, 1865.—BUSCH, F.: Ueber Fettembolie. Virchow's Archiv, Bd. 35, p. 321, 1866.—BERGMANN: Zur Lehre von der Fettembolie. Schmidt's Jahrbücher, Bd. 131, p. 316.—COHNHEIM: Untersuchungen über Embolische Processe. Berlin, 1872, p. 69.—CZERNY: Berliner Wochenschrift, Nos. 44 and 45, 1875.—RIEDEL: Deutsch. Zeitschrift f. Chirurgie, VIII., p. 571.—FLOURNOY: Contribution à l'étude de l'embolie graisseuse. Strasbourg et Paris. Virchow u. Hirsch, 1878, I., 226.—WIENER: Wesen und Schicksal der Fett-Embolie. Archiv für experiment. Path. und Pharm., Bd. XI., p. 275.—LÜCKE: Allgemeine Wien. med. Zeitung, 1880, p. 105.

Inasmuch as the occurrence of fatty embolism of the lungs is not even hinted at in *v. Ziemssen's* Cyclopædia, it would seem not out of place to describe the condition briefly here.

Zenker and *E. Wagner* were the first to discover that the smaller vessels in the lungs may become plugged with fat. The former attached no practical importance to the condition; but the latter was led to connect it with the origin of metastatic abscesses, and in his second publication on the subject expressed his surmise that sudden fatty embolism might be fatal through pulmonary hyperæmia and œdema, though he could bring forward no proof of this. We now know that *Wagner's* surmise was quite correct, and that many deaths after injuries which were formerly supposed to be due to shock, as well as death for which we were at a loss to account in some other affections, are in truth due to fatty embolism of the lungs.

There is reason to think that in almost every fracture of the long bones, at least, more or less of the medullary fat gets into the veins, and is carried into the circulation, but it is only very exceptionally that the quantity is sufficient to give rise to any symptoms or disturbance. Some of the fat may pass through the pulmonary circulation and be deposited in capillaries or small vessels elsewhere, especially if the action of the heart be feeble.

As a typical case I will cite that reported by *Bergmann* (*Berl. klin. Wochen.*, No. 23, 1873). A fall of thirty feet resulted in comminuted fracture of the thigh. After a short time complaint was made of pain in the chest, and later a frothy, bloody expectoration appeared, the respiration increased in frequency, the lips became livid, the temperature rose, and fine moist râles were heard throughout both chests. *B.* made the diagnosis of œdema of the lungs caused by fatty emboli, eliminating traumatic pneumonia, and pulmonary hemorrhage. Death occurred forty-nine hours after the accident. At the autopsy both lungs were hyperæmic, œdematous, dotted with small dark spots, and contained hemorrhagic infarctions the size of a pin's head.

Egli (Jahresbericht der gesammt. Med., 1873, p. 214) reports two cases of fat embolism in which the source of the fat was supposed to be thrombi in the right heart which had undergone puriform softening. The degenerated blood-clots were found to contain large and small fat-drops.

Riedel reports two cases of injury to the soft parts, in one of which fatty embolism occurred to a great, in the other to a slight degree. He also found it in three cases of inflammation of the bones, with and without subsequent operation on the same. In two cases of inflammation of the soft parts he found no trace of it.

Out of 250 bodies examined by *Flournoy* in the Pathological Institute at Strassburg, fatty emboli were found in 26 cases—about 10 per cent—but he throws out three of these as they were imperfectly reported. The remaining 23 cases he divides into two groups with reference to etiology; the first group containing 13 cases, the second 10. Of the first group there was injury to the bones in five cases, osteo-myelitis in one, acute suppuration in fatty tissue in two, suppuration in both bone and soft parts in five. Of the second group there was contusion of the soft parts in one case, but in the other nine there was no injury whatever, either to the bone or soft parts. Six of these, however, presented peculiar changes in the bone-marrow which *F.* considers the source of the emboli. The marrow, namely, was very soft, fluid, and dark in color; and microscopic examination showed that these changes were due to intense hyperæmia and marked atrophy. He does not attempt to decide whether the condition arose from hæmorrhage or from simple hyperæmia with laceration of the fat-cells and thus absorption. In support of his view that the hyperæmia of the marrow is the cause of the embolism he cites the case of an old person whose bone-marrow was atrophic, but pale in color, and in whom no embolism could be found. In three of his cases the condition was the sole apparent cause for death, and in many of the others no doubt contributed materially toward the fatal result. He has collected and analyzed 140 cases, including his own, and finds that in 18 of these—12.86 per cent—death was due to fatty embolism alone. Injury to the bones was responsible for 72 per cent of the cases, and one case was due to fatty softening of the brain.

Sanders and *Hamilton* (*Edinburgh Med. Journ.*, July, 1879) report a case of diabetes mellitus with death from fatty embolism. One morning dyspnœa came on, followed in the evening by unconsciousness, twitchings of the extremities, and death at four the next morning. Lipæmia was very marked and the embolism seemed to be largely confined to the lungs and kidneys, which were in other respects sufficiently healthy. No fat was found in the uriniferous tubes. It is probable that we have here the true cause of the sudden dyspnœa, coma, and death, which is not infrequent in diabetes, and which has been attributed to acetonæmia by *Kussmaul* and *Balthazar Foster* among others.

Fatty emboli in the lungs and other tissues may be detected by treating sections with perosmic acid, which turns the fat-drops a deep and uniform black without attacking the other structures.

Wiener has shown experimentally that the fat is absorbed into the circulation and eliminated with the urine by the kidneys.

HYPERÆMIA AND ŒDEMA OF THE LUNGS.

BIBLIOGRAPHY.

Köster: Virchow's Archiv, Bd. 55, p. 455.—Orth: Virchow's Archiv, Bd. 58, p. 126.—Strümpell: Acute Primary Fatal Œdema. Archiv der Heilkunde, 1877, p. 193.—Welch: Zur Pathologie des Lungen-Œdems. Virchow's Archiv, Bd. 72, p. 375.—Mayer: Bemerkungen zur experimentellen Pathologie des Lungen-Œdems. Prager Vierteljahrschrift, 1879, I., p. 141.—Lund: Acute Fatal Pulmo-

nary Œdema. Schmidt's Jahrbücher, 1879, IV., p. 29.—BORDEN: Œdema of the Lungs. With cases. Boston Med. and Surg. Jour, 1880, II., p. 9.

PATHOLOGY AND ETIOLOGY.

Welch and *Mayer* are led to conclude from experiments on dogs and rabbits that none of the explanations which have been current up to the present time as to the mode of production of acute general pulmonary œdema are really satisfactory.

The former author criticises the use which has been made hitherto of the term "collateral" hyperæmia and œdema, and remarks that two very different conditions have been included under it: A sharp distinction should be drawn between local serous transudation in the vicinity of inflammatory foci and new formations, and acute general œdema. He agrees with *Cohnheim* that the former depends on inflammatory changes in the walls of the vessels primarily; and only secondarily, and in small measure, on compensatory increase in the blood-pressure within the vessels of the part. General œdema, on the other hand, generally comes on suddenly and may as suddenly disappear; it affects both lungs, occurs in connection with a great variety of diseases, but is a constant accompaniment of none, and very often appears during the agony, an attendant rather than the cause of death.

Welch then ably discusses the causes of pulmonary œdema as laid down by *Niemeyer* in his Handbook of Medicine and *Hertz* in the Cyclopædia, showing that some of them are unsound and that neither individually nor collectively do they serve to explain the facts. His experiments were chiefly directed to the attempt to produce œdema from passive congestion in the lung, which latter may be brought about by obstruction to the circulation in the aorta, the pulmonary veins, or the left heart. He found that it was necessary to tie the aorta itself between the innominate and left subclavian, and all its branches except one—either one of the carotids or the right subclavian—before he could induce pulmonary œdema as a constant result; or, in other words, *that obstruction to the systemic circulation can give rise to pulmonary œdema only when such obstruction attains a degree scarcely conceivable in the human subject.* He then experimented on the pulmonary veins and found that *œdema did not result, nor was the pressure in the pulmonary artery materially increased, until the circulation was nearly completely shut off through all of them.* Occlusion of all the pulmonary veins of one lung was followed by infarction, never by œdema. He then turned to the left ventricle and found that he could often succeed in paralyzing it in the rabbit by pinching the wall between the fingers, while the right ventricle retained its activity for a time; and *general pulmonary œdema was the invariable result.* The right ventricle is more easily paralyzed than the left, owing to the thinness of its walls, but paralysis of this was never followed by œdema. From these considerations he forms the hypothesis that general pulmonary œdema is due to *predominant weakness of the left ventricle,* and then goes on to show that this hypothesis will explain the facts and account for the great rapidity with which œdema may appear and disappear and its remarkable inconstancy.

Mayer's experiments bear out those of *Welch*, but also throw some new light on the subject. He found that if the arteries ascending to the brain (innominate and left subclavian) are laid bare according to *Kussmaul's* method and occluded, non-curarized animals get severe convulsions and, in the great majority of cases, extensive pulmonary œdema. In curarized animals, treated in the same way in other respects, œdema does *not* occur. After closure of the above-named arteries, the œdema was so marked as not to require a post-mortem to prove its existence. The tracheal canula was filled with a reddish frothy fluid within one or two minutes, and this fluid was so abundant as to render prompt artificial respiration fruitless. Arterial tension is greatly increased by the anæmia of the brain, while the forcible respiration and muscular spasms certainly contribute materially to forcing the blood upwards to the right ventricle. In the curarized animal the muscular fibre is paralyzed, and consequently forcible respiration, spasms, and œdema are absent. In short, the conclusion is that those conditions which materially hinder the discharge of the blood from the left ventricle or the lungs bring about at the same time indirectly through the cerebral centres other conditions which increase the accessory onward pressure of the blood, and keep the right ventricle so fully supplied that stasis in and œdema of the lungs result.

ATELECTASIS.

BIBLIOGRAPHY.

SMITH: Med. Times and Gazette, 1873, March 22d.—KJELBERG: Behandlung der angeborenen Atelektase durch warme Wasserdämpfe. Jahrb. für Kinderheilkunde, N. F., VI., 61–65.—HAUKE: Wien. med. Presse, 1774, XV., 34–36.—ROSENBACH: Berliner klin. Wochenschrift, 1878, No. 12.—LICHTHEIM: Versuche über Lungenatelektase. Archiv für experiment. Path. und Pharmak., Bd. 10, p. 54.

ETIOLOGY.

Lichtheim produced atelectasis experimentally on rabbits by closure of a main bronchus with laminaria tents, by ligation, and by opening the pleural cavity. His results confirm the idea of *Virchow* that the air which is shut in by the closure of the bronchus is absorbed by the blood-vessels, and go to show that the elastic contractility of the pulmonary tissue continues to act until the air is completely absorbed.

He thinks his experiments show that atelectasis in connection with fluid in the pleural cavity is not always due to compression, as has been almost universally held. Very large effusions do, it is true, compress the lung and squeeze the air out of it; but a different explanation must be given in some cases of moderate serous effusion in which the pressure exerted on the lung by the fluid must be decidedly less than the ordinary atmospheric pressure, and yet that portion of lung immersed in the fluid is leathery and completely devoid of air, though not also of blood.

In these cases, that part of the lung which lies below the level of the fluid cannot follow the inspiratory enlargement of the thorax, and the air which it contains, not being renewed, must necessarily be *absorbed.*

If this view be correct, the number of cases of atelectasis by compression is much smaller than has been supposed.

TREATMENT.

Kjelberg, of Stockholm, recommends a new method which he has employed in three cases of congenital atelectasis. A wooden frame was placed over the bed and covered with blankets so as to form a sort of tent, and this was converted into a steam bath by keeping a vessel of hot water in it constantly, the water being renewed every half-hour. The temperature was thus kept at 26°-30° C. and the treatment continued during eight to fourteen days. As the respiration and general condition improved, the temperature was gradually lowered and the coverings removed, but the air of the room was still kept moist for some time. *Hertz* tried this method in one case, but had no great success with it, and truly remarks that the difficulties in the way of carrying it out thoroughly are not inconsiderable.

The last-named author reports favorably on the use of compressed air, especially during the stage of absorption of pleuritic effusion (second edition of *v. Ziemssen's* Encyclopædia). "I have repeatedly seen this method exert a favorable influence, not only in aiding the re-expansion of the lung and thus diminishing the risk of permanent deformity of the chest, but also in promoting rapid absorption of the fluid. It has been attended with specially happy results after thoracentesis. Expiration into rarefied air, should, however, be combined with the inspiration of compressed air to a certain extent, in order to avoid undue dilatation of the sound tissue. For when a considerable portion of lung is compressed the healthy portions are liable to be the seat of collateral hyperæmia, with œdema and consequent loss in elasticity; and these parts will offer less resistance to the increase in pressure of the inspired air and be in danger of becoming permanently emphysematous." This method is to be preferred to *Hauke's* pneumatic cuirass, which is said to exert eccentric force on the parietes and lungs when it is applied to the chest, and the air between the body and the cuirass is rarefied. It is true that *Hauke* recommends his procedure especially for asphyxia in new-born infants and collapse of the lung in children, cases in which co-operation on the part of the patient is, of course, out of the question.

EMPHYSEMA.

BIBLIOGRAPHY.

J. LANGE: Ueber das substantive Lungenemphysem und dessen Behandlung mit comprimiter Luft, 1870.—WALDENBURG: Die lokale Behandlung der Krankheiten der Athmungsorgane, 1872.—THIERFELDER: Pathol. Histologie der Luftwege, 1872.—IMMERMANN: Correspondenzblatt der Schweizer Aerzte, 1873, No. 1.—LANGE: Die pneumatischen Cabinette und die transportablen pneumat. Apparate. Med. Central-Zeitung, 1874, Nos. 28-30.—HÄNISH: Deutsch. Archiv f. klinische Med., 1874, Bd. XIV., p. 445.—GEYER: Diss. Jena, 1874.—SCHNITZLER: Wien. med. Presse, 1874, Nos. 14, 15, 17, 18, 22, 23. Wiener Klinik, Jahrg. I., 1875, Heft 6.—STÖRK: Mittheilungen über Asthma bronchiale und die mechanische Lungenbehandlung. Stuttgart, 1875.—GANGHOFNER: Prager Vierteljahrschr. f. pract. Heilkunde, Bd. IV., p. 79.—WALDENBURG: Die pneumatische Behandlung der Respirations- und Circulationskrankheiten, Berlin, 1875.—BIEDERT: Deutsch. Archiv f. klin. Med., 1876, Bd. XVII., p. 165, Bd. XVIII., p. 115.—COHEN, J. S.: Therapeutic Uses of Compressed and Rarefied Air. Transact. Coll. Physicians of Philadelphia, 1876, p. 165.—POWELL: On Some Effects of Lung Elasticity in Health and Disease. Medico-Chirurgical Transact., 1876, vol. LIX., p. 165. EPPINGER: Das Emphysem der Lungen. Prager Vierteljahrschrift, 1876. KNAUTHE: Handbuch der pneumatischen Therapie. Leipzig, 1876; Bericht über pneumatische Therapie. Schmidt's Jahrbücher, 1878, I., p. 135.—BERKART: On Asthma. London, 1878.—SCHNITZLER: Asthma. Wiener med. Presse, 1880.

HAY ASTHMA.

BIBLIOGRAPHY.

BLACKLEY: Cause and Nature of Hay Fever. London, 1874; New Observations on Hay Fever. London, 1877.—MORRILL WYMAN: Autumnal Catarrh. Boston, 1876.—BEARD: Hay Fever. New York, 1876.

THEORY OF THE DEVELOPMENT OF EMPHYSEMA.

That forcible inspiration may be a cause of emphysema is shown by *Waldenburg's* pneumatometric investigations. He found that in bronchitis and asthma inspiration is normal, while expiration is deficient; the air is thus expelled through the narrowed bronchial tubes only imperfectly, and carbonic acid accumulates sufficiently to increase the demands of the system for oxygen. This demand is partially satisfied through forcible inspiration, but at the same time more air enters the alveoli than can be expelled, and thus dilatation of the alveoli, loss of elasticity, and emphysema are induced.

Hertz thinks it very doubtful whether increase in the inspiratory force alone can produce emphysema in healthy lungs with unimpaired expiration; for it is reasonable to suppose that the increased pressure is, under these circumstances, so uniformly distributed throughout the lungs as to be incapable of doing mischief. According to *Waldenburg*, unwonted calls upon the respiratory organs, as in much mountain or stair climbing, may cause emphysema, and the author cites the case of a medical student in support of this view. The young man came from a small place where the houses were low to Berlin, and there took a lodging in the fifth story; he became emphysematous, apparently from mounting the four flights rapidly several times every day. *Pepper* (*Phila. Med. and Surg. Reporter*, April, '74, p. 373) thinks that prolonged and severe exercise of the muscles of the arms and shoulders, such as that to which some classes of laborers are subjected, produces emphysema through increase in the expiratory pressure; but *Hertz* regards this explanation as incorrect, and remarks that in cases of this kind there are probably primary nutritive changes in the lung tissue which act as predisposing causes, though their existence has not as yet been proved.

Hertz expresses the opinion, furthermore, in the second edition *v. Ziemssen's* Encyclopædia, p. 456, that the inspiratory as well as the expiratory act plays a part in the production of emphysema; the former in the diffuse form of the disease, the latter in those cases in which it is localized chiefly in the upper portions of the lungs. In the diffuse form, both acts may, indeed, contribute to the result; in chronic bronchitis, for instance, increase in the force of inspiration tends to produce general dilatation of the alveoli, while the upper lobes bear the brunt of the heightened expiratory pressure incident to the attacks of cough.

Whether asthma plays the part of cause or of effect may, in individual cases, be very difficult to determine. There is no doubt that asthmatic attacks intensify pre-existent emphysema, and that, vice versa, emphysema and the circulatory disturbance which it involves, favor the occurrence of asthma, the two having a very close relation to one another. The pneumatometer shows deficient expiratory power in cases of asthma, without previous emphysema, precisely as in emphysema itself.

This latter fact speaks in favor of the views of *Berkart*, who most vigorously and ably maintains that asthma is not a disease at all, but "*only one link in a chain of quasi-independent affections, which commences with changes of the pulmonary tissue, and terminates with emphysema or bronchiectasis*"—in fact, a symptom, indicating always structural modifications of the lung tissue (see a review of *Berkart*, *Boston Med. and Surg. Journ.*, Feb. 12th, 1880).

PROGNOSIS.

The experience of the last few years in the treatment of emphysema shows that some cases may be even cured which were formerly supposed to be incapable of improvement, though this remark holds true only of cases in which there is some impairment of the elasticity, but no extensive rarefaction of the lung.

It has been shown of late that the pneumatometer is of great service in prognosis, affording us information, not only as to the degree of existent deficient ins- or expiratory power, but also as to the value and effect of our therapeutic measures. Increase in the pneumatometric measurements and simultaneous improvement in the condition and symptoms of the patient are of favorable prognostic import, while non-increase or diminution are correspondingly unfavorable. The spirometer is also used by *Waldenburg* for purposes of prognosis; he finds that it is a rather unfavorable sign when the capacity of the lungs is diminished as much as fifty per cent—in men to 1,500–2,000 cc., in women to 1,000–1,500; but absolutely unfavorable if it be as low as 1,000–1,400 in men, and 700–1,000 in women. It is, however, to be borne in mind that patients differ greatly in the skill with which they use the spirometer, and we should never, therefore, be content with the results of one trial, but repeat it several times on different days before drawing our conclusions.

Assistance in prognosis may also be derived from the use of the pneumatic apparatus with a view to the treatment of emphysema. It is of good import, showing that the thorax is distensible, and the lung still retractile, if an amount of air can be drawn out of the lung equal to the normal vital capacity; whereas with less amounts the contrary is true. We can always, hence, be sure that there are textural changes in and atrophy of the pulmonary tissue, for the repair of which there are, at present, no means in our power, when the spirometer shows small vital capacity, the pneumatometer low measurements, and at the same time the amount of air extracted by the pneumatic apparatus is relatively deficient. The worst cases are in persons advanced in years with ossified and fixed thorax.

DIAGNOSIS.

These same instruments are also of service in diagnosis.

The spirometer indicates, as in various other affections of the respiratory organs, the degree of diminution in the vital capacity; but does not enable us to distinguish between these affections. The pneumatometer, on the other hand, shows that in emphysema and bronchiolitis the expiratory force is diminished, while in phthisis the loss is in inspiration, and we are enabled to detect commencing emphysema with the aid of this instrument in people who appear quite well, complain of nothing, and are only slightly short of breath on exertion. *Waldenburg's* careful investigations show also that we can determine the degree of the affection. In slight cases, in which dyspnœa is present only after unusual effort, and there is no modification of the percussion note, it is found that the expiratory pressure is somewhat less than the inspiratory, instead of being decidedly greater, as it is under normal conditions. In a later stage of the disease, when slight exertion suffices to induce dyspnœa and percussion reveals unquestionable pulmonary dilatation, it is found that the expiratory is one-third or one-half less than the inspiratory pressure; the latter remaining usually normal in amount, though it may be increased in order to compensate for the deficiency of the former. In the worst cases, finally, with bronchitis of years' standing, severe dyspnœa and asthma, cyanosis, œdema, and rigid thorax, the inspiratory pressure is found to be diminished also, though it still remains greater than the expiratory.

The stethograph also affords valuable information, *Riegel's* observations showing that expiratory insufficiency is indicated by very characteristic curves which deviate from the normal standard in proportion with the emphysematous changes in the lungs. The normal curve is represented during inspiration by a gentle wavy rise with gradually increasing rapidity, and then gradual descent; during the transition from in- to expiration, by a line curved in the form of a bow which then descends with uniform rapidity until the respiratory pause. The curve of emphysema, however, has a much less gradual and an unduly rapid ascent during inspiration, and falls away at an acute angle during expiration, which latter is further characterized by increased duration and a rapidity which is at first normal, gradually decreases, and in the last third becomes very much less than it should be.

The strikingly short in- and prolonged expiration, as contrasted with the nearly equal duration of the two acts in a healthy person, is very characteristic of emphysema, and *Riegel* holds that the change in their relations is exactly proportional to the loss of pulmonary elasticity. The lung which is already distended cannot take in much more air, but does its best with the aid of the accessory muscles of respiration. The enlargement of the chest is not gradual as in healthy persons, but inspiration is rapid and brief, as is indicated by the steep rise in the curve, and the first portion of expiration is rendered relatively rapid by what elasticity is still retained by the lung, the pressure of the abdominal organs, and the tendency of the ribs to return to a position of rest. The second portion of expiration is more slow; and the last portion the slowest of all, for the reason that all save the accessory forces of the act have been exhausted during the first period, and these latter work feebly and at a disadvantage against the resistance of the chest-walls.

TREATMENT.

According to *Berkart*, the nutritive changes in the pulmonary tissue which are in the course of time followed by emphysema and asthma, are in the great majority of cases traceable to chronic bronchitis, and catarrhal pneumonia complicating whooping cough, measles, and typhoid fever; it may hence be in our power by careful treatment of these affections to save persons from much future misery. Whether this view be correct or not time will show; but, if we err at all, we shall certainly err on the safe side in attempting to keep under and cut short as far as possible those affections in which inflammation of the bronchi has a tendency to spread to the parenchyma of the lung. Nor is this all; careful attention to hygienic measures should also be enforced after the symptoms of these affections have subsided, that the general nutrition may be raised to the highest possible point, and the lung tissue thus restored to a perfectly healthy condition. Cases in which an hereditary tendency to emphysema and asthma is discovered should, of course, engage our most earnest attention. Asthma having been generally considered hitherto as dependent on derangement of the central nervous system, treatment has been directed almost exclusively to the palliation of the dyspnœal paroxysm, and the underlying condition has been greatly neglected. It should be our object to arrest the progress of existing pathological lesions and to maintain the healthy portion of the lung in a state of greatest efficiency; the proper way to attain this object being to improve the nutrition of the body in general, and indirectly that of the lungs, and to restore the normal function of the bronchial surface.

Fresh air, sunlight, warm clothing, cleanliness, suitable exercise, and careful attention to the gastric and intestinal digestion, are the most important points. Proper ventilation of the sleeping room is of prime moment.

There are certain patients who are so afraid of fresh air that its effect on them is almost like that of alcohol. The chemical changes of the body which have been hitherto at the lowest ebb are roused by a free supply of oxygen to an energy previously unknown, at times even so great as to produce an almost febrile condition. Moreover, by prolonged confinement in impure air, the bronchial mucous membrane is apt to be anæsthetized by the narcotic influence of the carbonic acid; then, on exposure to a pure atmosphere, the membrane recovers part of its sensibility, and cough is readily induced. But this return of bronchial sensibility is not regarded by these patients as a favorable sign, rather as a "fresh cold" and they refuse to continue the treatment, returning to narcotics in order to suppress the cough. Hence, the longer they have breathed in a vitiated atmosphere, the more gradually are their habits to be changed; otherwise the neglect of this precaution would deprive the sufferers of the benefit of the treatment, and cause the practitioner the annoyance of seeing his best efforts frustrated (*Berkart*). Sufferers from hay asthma or autumnal catarrh cannot, of course, afford to fight out their annual attack, but must, if it be possible, take refuge in some spot which gives them immunity from their enemy. [Reference is made in the bibliography at the beginning of this article to the most important recent monographs on this subject.]

The medical literature of the pneumatic treatment since the publication of the first edition of *v. Ziemssen's* Cyclopædia is enormously copious, but there is not much which is really new. *Waldenburg's* apparatus has been modified by *Schnitzler* and others, but the underlying principles remain the same. *Schnitzler* has, for instance, constructed an apparatus—it is called portable—consisting of twin cylinders; one of these contains compressed, the other rarefied, air; and the patient can thus change readily from one to the other, economizing his own time as well as that of the physician.

In the second edition of *v. Ziemssen's* Encyclopædia, *Hertz* has greatly modified and enlarged his remarks on the pneumatic treatment as contained in the first edition; and as it is not likely that any further change of consequence would be made if a third edition were to appear now, I shall simply translate a short portion of his exposition.

The favorable results which have been obtained by the use of the pneumatic apparatus these last five years ('72–'77) show that in it we have a valuable means of alleviating and curing emphysema. According to *Waldenburg*, expiration into rarefied and inspiration of compressed air are indicated in all cases of obstinate bronchitis, in which the pneumatometer indicates expiratory insufficiency, whether there be any other sign of emphysema or no. In case the pneumatometric measurements do not increase at all after several weeks' use of the apparatus, or if the thorax be rigid and we are satisfied that there is already extensive atrophy of the parenchyma of the lung, the treatment is to be discontinued, as it only serves unnecessarily to fatigue the patient. The pneumatic treatment is, moreover, contra-indicated if there be secondary degeneration of the heart.

The effects of the pneumatic apparatus in emphysema are then briefly as follows:

By *expiration into rarefied air:* 1. An increased amount of residual air and carbonic acid are sucked out of the lung, and consequently more oxygen can be absorbed and pulmonary ventilation is furthered. 2. The lung is diminished in size more than after an ordinary expiration. 3. This diminution becomes gradually permanent, as is evidenced by percussion and direct measurement; and 4. Simultaneously with the gain in elasticity of the lung and the diminution in the residual air, the vital capacity is increased.

Inspiration of compressed air undoubtedly leads in time to dilatation of the lungs, and thus acts directly contrary to the above treatment unless it be practised with the greatest caution; with this proviso, however, it is unquestionably of service in emphysema, inasmuch as it tends to counteract hyperæmia of the bronchial mucous membrane, whether this hyperæmia be already present and intensified, or called forth by the use of the rarefied air. Compressed air also increases the force of the cardiac contraction and heightens the pressure in the systemic circulation, as is shown by the pulse becoming harder, and thus relieves the pulmonary circulation; it may, furthermore, act as an expectorant in bronchitis, the aërial current dislodging mucus impacted in the finer tubes. In the treatment of emphysema, consequently, a subordinate and symptomatic position alone can be accorded to the inspiration of the compressed air, whereas expiration into rarefied air is to be regarded as directly curative.

As for medicinal treatment, *Germain Sée* (*Gazette Méd. de Paris*, 1878, p. 69) uses iodide of ethyl and iodide of potassium in asthma. The former is given by inhalation during the paroxysm, in a dose of five ten drops and, it is said, with very satisfactory results. The potash is given in the intervals between the attacks to ward off their occurrence. He gives twenty to forty-five grains per diem and is not afraid of iodism which he combats by increasing the dose of the drug. In twenty-four cases treated in this way, which have been under observation for several years, the results are very satisfactory.

Dr. *J. P. Oliver*, of Boston, has also used large doses of iodide of potash in asthma of late with surprising success (*Boston Med. and Surg. Jour.*, Feb. 19th and March 4th, 1880). He increases the dose gradually up to fifty or sixty grains per diem and continues it for a long time, warning the patient not to expect relief too soon and that six weeks may elapse before any change can be noticed. Some of his patients were unable to take the iodide, even in small doses, and he then substitutes hydriodic acid. The syrup of hydriodic acid prepared by Robert Gardner, of New York, he considers the best form of administration, it being agreeable to the taste and not very likely to be affected by exposure to air and light. He begins with small doses, twenty or thirty drops well diluted with water, and taken half an hour to an hour before meals; if taken after meals it may disturb the stomach. The dose is gradually increased, but should not exceed a tablespoonful. In cases of chronic bronchial catarrh, and in fact in all cases where iodine is indicated, he has found this syrup of great value. The above is taken from a mere preliminary notice of the method of treatment, a more detailed account of which, with a large number of cases, is to appear in the *Boston Medical and Surgical Journal* shortly.

Westbrook (*Proceedings Med. Soc. Kings Co., N. Y.*, 1879, IV., p. 7) reports ten cases of asthma with bronchitis and emphysema, all of which were greatly relieved by iodide of potash which he usually gives in seven and a half grain doses four times a day.

It should be mentioned that iodide of potassium is contra-indicated when the bronchial mucous membrane is acutely inflamed; the iodine in contact with the atmosphere being disengaged from its combination with the albumen of the blood, and when thus set free being apt to irritate the surface of the air passages.

Penzoldt (*Berlin klin. Wochenschrift*, No. 19, 1879) first called attention to the value of quebracho bark—from a Brazilian tree—as a remedy for dyspnœa, whatever its cause apparently. It is said that South American physicians attribute antifebrile qualities to the remedy, similar to those of quinine. *Penzoldt* could see no effects from the drug when used as an antipyretic, but found that the alcoholic extract of the bark dissolved in water had a marked effect on dyspnœa, decreasing the frequency of and greatly facilitating respiration. He gave the drug to hospital patients with emphysema (both simple and complicated by bronchitis), phthisis, chronic pneumonic processes associated with periodic attacks of an asthmatic nature, pleurisy, etc., and concludes that "we possess in quebracho bark a remedy which affords more or less complete relief for hours from the various forms of dyspnœa incident to diseases of the organs of respiration and circulation; without, at the same time, producing any sort of disturbance. Its action is manifested by decrease in the frequency, and often in the depth, of respiration, diminution of the cyanosis, and, above all, improvement in the subjective condition of the patient."

Berthold and *Picot* (*Berl. klin. Wochenschrift*, No. 52, 1879) also report on the drug. The former found relief from it in only two out of six cases of phthisis. The latter administered it in dyspnœa from catarrhal pneumonia, asthma, and valvular disease of the heart—of each one case—with decidedly beneficial effect. He then took it himself while on a vacation tour in the mountains and was satisfied that the drug enabled him to walk up hill with much less fatigue and shortness of breath.

This is, of course, far too slender a basis of facts to enable us to decide on the merits of the remedy, and it is more than possible that further investigation will consign quebracho to the place occupied by so many of the drugs of the pharmacopœia, but no harm can apparently come from giving it a fair trial. The writer is informed by a competent authority that the bark is not at present to be had in this country, but that it will be on hand before this volume appears in print.

Since the above was written, a new report has come in from *Laquer* (*Wiener med. Presse*, Jan. 25th, 1880). He administered the remedy according to the following formula:

℞ Ext. corticis quebracho,	12.
Mucilag. gum acac.,	40.
Aquæ,	200. M.

to twelve patients with emphysema and severe bronchits. In some cases decided relief followed a dose of one to two teaspoonfuls, but in others no effect was observed. He tried it also in one case of valvular disease of the heart, but it seemed to aggravate the sufferings of the patient, and he did not feel encouraged to give it to other cases of the kind.

With a view of ascertaining whether the remedy produced permanent or only temporary relief, it was then administered in doses of one to two teaspoonfuls thrice daily in nine cases of emphysema, one of chronic bronchitis, and one of right pleurisy. The results were not unsatisfactory, though not as good as had been hoped for. In some of the cases the drug caused, after a few days, headache with a sensation of heat and confusion in the head and giddiness, these symptoms increasing so that the patients begged to be let off.

Conclusions.—1. The drug appears to be a useful palliative in many cases of dyspnœa in emphysema and chronic bronchitis; but in other cases, especially in old people, is of no service.

2. After prolonged administration, various disagreeable effects may appear, rendering it necessary to omit the medicine.

3. The frequency of respiration is diminished with a certainty proportional to its previous increase above the normal standard. The remedy has no constant effect on the pulse.

GANGRENE OF THE LUNG.

BIBLIOGRAPHY.

Traube: Berliner klin. Wochenschrift, 1871, No. 25.—Louisa Atkins: Ueber Gangräna Pulmonum der Kinder. Dissert., Zürich, 1872.—Lancereaux: Archives Générales de Méd. Mars, 1873.—Foot: Dublin Jour. Med. Sciences, Jan., 1873. —Zuchner: Ueber Lungenbrand. Dissert., Berlin, 1874.—Bucquoy: L'Union méd., 1875, Nos. 84–95. Bull. gén. de thérapeutique, 15 Août, 1875. Gazette des Hôpitaux, 1879, p. 738.—Hanot: Progrès méd., 1876, No. 4.—Refer: Med. Centralzeitung, Mai, 1876.—Huntington: Boston Med. and Surg. Journ., 1876, II., p. 485. Remarks on Thirty-two Cases of Pulmonary Gangrene.—Filehne: Ueber die Vorgänge bei dem Lungenbrande, etc., etc. Schmidt's Jahrbücher, 1877, Bd. 185, p. 33.—Kersten: Ueber Lungenbrand, Berlin, 1878.—Curschmann: Zur Lokalbehandlung der putriden Bronchial- und Lungenaffectionen. Berliner klin. Wochenschrift, 1879, Nos. 29 and 30.

ETIOLOGY.

In the second edition of *v. Ziemssen's* Encyclopædia, *Hertz* adds that diseases or conditions which involve very great prostration with degeneration of the heart and sluggish circulation may result in thrombosis in the systemic veins, infarction, and gangrene of the lungs. *Steffen* suggests the possibility of thrombosis of the vessels of the lung in consequence of feeble and superficial respiration. Some English writers have supposed the existence of what they call "gangræmia"—a special condition of the blood favorable to the occurrence of gangrene—but *Hertz* knows of no case which does not admit of some more satisfactory explanation. He cites five cases in which blows and falls on the chest or shoulders have been followed by gangrene—traumatic—and a case is reported by *Wahl* (*Schmidt's* Jahrb., 1878, Bd. 178, p. 32) of a man who received a violent blow on the head, laying bare, though apparently not fracturing the skull. The patient kept his bed for five days without an unfavorable symptom, but then, his wound being healed, he rose contrary to advice and began to work. Almost immediately he had a chill followed by pain in the region of the right nipple, gangrene of the lung, and death.

Magrath (*Lancet*, 1880, I., p. 89) reports a case of the passage of a head of grass into a bronchus with death after twelve weeks. The grass was found in the lower right lobe, about one-third of which was gangrenous. The diaphragm was perforated, the upper surface of the liver showed commencing softening, and the right side of the bodies of two dorsal vertebræ were excavated and carious. There are a number of similar cases on record, but it is on the whole surprising how large a proportion of the foreign bodies which get into the lungs are sooner or later expelled with final recovery on the part of the patient.

Hertz states, but without quoting his authority, that gangrene of the lung has been noted with special frequency during epidemics of diphtheria. Nothing is definitely known as to the relations of the two diseases, but the idea is suggested that, in those cases especially in which they are co-existent, the gangrene is really to be regarded as pulmonary diphtheria caused by low organisms.

Lancereaux has found bacteria in the gangrenous fluid and in the blood after death; he inoculated a rabbit with a very small quantity of the latter, causing death in twenty-four hours, and found precisely similar organisms in the animal's blood.

DIAGNOSIS AND PROGNOSIS.

Traube and *Leyden* consider the absence of elastic fibres in the sputum as very characteristic of grangrene as contrasted with other affections which involve destruction of the pulmonary tissue.

Filehne thinks that the statements of the above-named authors are too strong, though he allows that the elastic fibres are often absent, or present in far less numbers than one would naturally expect. He was led to experiment with a view to explaining this fact, and his results seem to show that gangrenous sputum or pulmonary fluid contains some ferment which has the property of dissolving albumen and elastic tissue, while it does not attack fibrous tissue.

Bucquoy thinks that by observance of the following points a diagnosis may be often made before the sputum becomes offensive. As was first remarked by *Stokes*, the pain in the side is far more obstinate and severe than in any pleurisy or pneumonia; the temperature and pulse rate are not increased in proportion to the general phenomena of the disease, while dyspnœa and prostration of the system are very extreme. It is conceivable, of course, that the diagnosis should be reached from these symptoms alone, especially if the gangrene be diffuse, but we know that when it is circumscribed the disease may be far removed from the surface of the lung, and that the symptoms may not be very marked at first. On the other hand, the fever may be very intense in either form.

Huntington has collected thirty-two cases from the records of the Massachusetts General Hospital, being all the cases of the kind admitted from 1857 to 1875 inclusive. The results being brief and decidedly more favorable than leading authorities would allow us to expect, they are given in full. Seven cases were discharged well; six much relieved (these with proper care and favorable circumstances doubtless proceeded to ultimate recovery); three cases were temporarily relieved; five were not relieved, two of which were in the hospital but a brief period and were not treated; eleven cases terminated fatally.

COMPLICATIONS.

Cases with complication,	10
Cases without complication,	22
Phthisis as a complication occurred in	8

Emphysema as a complication occurred in	1
Cancer of the œsophagus as a complication occurred in . .	1

In seven of the cases without complication gangrene followed pneumonia, and was diffuse. Leaving them out of the discussion, the following statement can be made. Of the fifteen cases without complication, five were well, six much relieved, one was relieved temporarily, three not relieved, none died. In other words, among the cases of *circumscribed gangrene* there were eleven favorable and four unfavorable terminations. On the other hand, of the complicated cases, four died, two were not relieved, two were temporarily relieved, and two were cured of gangrene. That is, two terminated favorably as far as the disease under consideration is concerned, and eight terminated unfavorably.

Summary of results and complications.

CASES WITHOUT COMPLICATIONS.

Terminated favorably, ...	73.3 + per cent.
" unfavorably, ...	26.6 + " "

CASES WITH COMPLICATIONS.

Terminated favorably, ...	20 per cent.
" unfavorably, ...	80 " "

TREATMENT.

Bucquoy recommends highly the tincture of Eucalyptus globulus in two-gramme doses, finding that the drug has a very marked effect on the fetor of the breath and sputum, and also alleviates the cough.

Draper (*Boston Med. and Surg. Journ.*, 1876, II., p. 595) reports the successful use of salicylic acid in ten-grain doses, thrice daily.

Wys has used creasote by inhalation with benefit, a few drops being poured on flannel, and prefers it to turpentine, as being less disagreeable and irritating.

Curschmann has employed since 1871, in private and hospital practice, a mask which covers the mouth and nose and resembles in appearance the mouth-piece of *Waldenburg's* pneumatic apparatus. A compartment in the mask or respirator is filled with sponge to receive the remedy—turpentine, carbolic acid, thymol, creasote—and the apparatus is recommended in all pulmonary affections characterized by fetid breath and expectoration. His experience with gangrene seems to be very slight, the cases being chiefly of putrid bronchitis and bronchiectasis. Carbolic acid was used in very strong alcoholic solution and also *pure* after having been liquefied by heat, giving rise to no unpleasant symptoms even when the respirator was worn for days; thymol was always used in alcoholic solution. The solution of either remedy has the advantage that the drug is thus more volatile, but the disadvantage, on the other hand, of being more irritating, and hence more likely to excite cough. The fetor was sometimes greatly diminished within twenty-four hours, though usually a somewhat longer period was required, and in many instances was entirely relieved; fever dependent on decomposition in the secretion ceased, and the bodily weight came up. Turpentine and creasote were used in the pure state, and the latter is preferred in cases with a tendency to hæmoptysis. It would seem worth while to try this method of treatment in some cases of circumscribed gangrene.

Two cases have been reported recently in England in which gangrene of the lung was treated by incision. Both were very severe and terminated fatally, though the operation afforded marked relief. The first of these cases is reported by *Cayley* (*Lancet*, 1879, I., p. 440). A man of forty entered the Middlesex Hospital, having been ill five weeks, and had fetid cough and expectoration two weeks. Prostration and emaciation were extreme, and small quantities of brownish, horribly offensive mucus were raised with great difficulty. The signs pointed to consolidation at the right base, but it was inferred that there was a full cavity at the spot, and an exploratory puncture was made with an aspirator needle. A few drops of fetid pus coming out, an incision three inches long was made in the ninth interspace, on a line with the lower angle of the scapula. About five ounces of frightfully offensive pus and several bits of gangrenous lung-tissue escaped; a drainage tube was inserted, and the cavity was washed out twice daily. The patient experienced great relief, ceased to cough and raise fetid mucus, the bad smell disappeared, and the temperature, which had been very high, fell to the normal point. He died of prostration five days after the operation. At the autopsy it was found that the right lower lobe was consolidated by pneumonia and firmly adherent to the chest-wall. The point where the cavity was most superficial was more than an inch from the surface of the lung.

In the second case, *Smith* (*Lancet*, 1880, I., p. 86), gangrene supervened on right pneumonia in a man of over sixty years of age. His condition became most critical and a cavity was detected in the right middle lobe. On consultation, it was decided that the only chance for life lay in opening the cavity and evacuating the contents, especially as the discharge was not free through the bronchial tubes. An aspirator needle was introduced to the depth of three or four inches near the angle of the scapula; no fluid escaped, but there was a gush of very foul air, and, on holding a candle near the open canula, the flame was blown to and fro with respiration. The canula was then used as a director; a knife was passed in between the ribs, and by its side a pair of dressing forceps, by opening which the wound was dilated sufficiently to allow of the introduction of a drainage-tube. Through this a little carbolic lotion was injected, which seemed to excite a coughing-fit and the forcible expulsion of half a pint of fetid pus. For the first week after the operation the improvement was very decided, but a change for the worse then set in and the patient gradully sank. There was no autopsy. Mr. *Smith's* conclusions are: "(1) When the opening through the bronchi seems to be insufficient as an exit for the fluid, or the passage of the gangrenous ichor seems to be setting up irritation in the bronchial mucous membrane; (2) the patient appears to be sinking rather than rallying; and (3) auscultation shows the presence of a cavity, an incision with a view to drainage is justifiable."

NEW GROWTHS IN THE LUNGS.

BIBLIOGRAPHY.

HÄRTING AND HESSE: Der Lungenkrebs—die Bergkrankheit in den Schneeberger Gruben. Vierteljahrschrift für Gerichtliche Medicin, 1879, Apr., July, Oct.—SCHMIDT'S JAHRBÜCHER: 1879, II., p. 81 et seq.—FINLAYSON: Primary Epithelioma of the Lung. Medico-Chirurg. Trans., Vol. LX., p. 313.

Finlayson adds three cases—one of his own—to the short list of observations of primary epithelioma of the lung, and many cases of the more common varieties of new growths in the lungs have been reported since 1874. The only article which requires especial reference is, however, that the title of which heads the above bibliography.

At Schneeberg, in south-western Saxony, there is a large cobalt mine, affording employment in 1877 to five hundred and eighty-four persons in all, and it has been known for some time that the workmen at this mine were especially liable to a peculiar thoracic affection, the nature of which the painstaking investigations of our authors go far to explain. Until seventeen years ago, the disease was regarded simply as consumption. The arrangement of the article is so involved and lacking in clearness that it is a difficult matter to extract from it a clear statement of the disease. I will, therefore, begin by stating their conclusions, and then enlarge upon them somewhat.

1. The disease endemic in the Schneeberg mines is pulmonary cancer and is the cause of about seventy-five per cent of all the deaths among the employees.

2. The disease assumes the form of lymphosarcoma, though in rare cases of endothelial carcinoma; both forms are, however, intimately connected, and originate invariably in the bronchial glands.

3. All of the miners who do not die from some accident incident to their calling or from some intercurrent affection, as it were, finally fall victims to cancer of the lung.

4. The disease never appears earlier than twenty and rarely later than fifty years after the occupation was embraced. The miners, especially those who are continuously employed in the drifts, are affected sooner than the masons and timber cutters, who do not pass their whole time under ground, and are occasionally furloughed.

5. A low condition of the general nutrition and previous affections of the lung or pleura, and emphysema, are all predisposing causes.

6. The immediate cause of the disease is arsenic, which in this mine is found uncombined with sulphur in the regulus of cobalt; is inspired; passes into the lymphatic current; is arrested in the bronchial glands, and there sets up an irritation which eventuates in sarcoma.

In most arsenic-bearing mines that metal is combined with sulphur, and the sulphide, being insoluble, is vastly less poisonous. The workmen in the mines of Sweden, Hungary, and Tyrol are said to be free from any disease of this nature, and there is little doubt that the depth of the mine and the difficulties in the way of ventilating it are not without a certain influence.

The anatomical appearances are shown by twenty autopsies to be briefly as follows:

The cutaneous veins of the neck, chest, and arms, especially on the affected side, are generally unduly prominent. The mucous membranes are pale, often livid, and the nails are almost always incurved. The intercostal spaces are broad, but the affected side of the thorax is rarely notably enlarged. The pleura of the side which contains the tumor is distended by a moderate effusion, which is sometimes blood-stained, and adhesions are always found at the root of the lung. The affected lung is diminished in size, sometimes very markedly, and the tumor is generally unilateral, yellowish-white in color, in some cases confined to the root of the lung, in others involving one or even both lungs, the pleura, mediastinum, pericardium, etc., and of large size. The disease may extend to the ribs or sternum, and secondary nodules of larger or smaller size are often found in the liver. No constant changes are found in any of the other organs which can be connected with the affection under consideration. The nodules are found only in the lungs in some cases, and when present in other organs are always more recent; tubercles have never been observed. The disease starting as a growing tumor at the root of the lung, the symptoms are usually very ill-defined at the commencement, and in some cases never assume any prominence. Later they vary of course according to the direction taken by the growth and the parts compressed by it, though a general cachexia is well marked. The duration of the disease, dating from the time when medical aid is first invoked, varies usually between six and eighteen months, though in one case three years elapsed; and at the autopsy a fattily degenerated tumor the size of a hen's egg was found at the root of the lung.

Treatment has hitherto proved practically of no avail.

Out of an average total of 650 employees, the disease has proved fatal as follows:

1869–1871,	63 cases.
1872–1874,	47 "
1875–1877,	40 "

The authors claim only approximate accuracy for these figures and attribute the diminution in the number of cases of late to the adoption of hygienic measures, especially to improved ventilation of the mine.

PULMONARY CONSUMPTION.

BIBLIOGRAPHY.

Pidoux: Etudes générales et pratiques sur la phthisie, 1873.—Massini: Ueber die Heilbarkeit der Lungenschwindsucht. Deutsch. Archiv für klin. Med., 1873, Bd. XI., p. 446.—Sommerbrodt: Ueber die Abhängigkeit phthisischer Lungenerkrankungen von primären Kehlkopfaffectionen. Arch. für experimentelle Pathologie, Bd. I., 1873.—Mosler: Ueber lokale Behandlung von Lungencavernen. Berliner klin. Wochenschrift, 1873, No. 43.—Bennet: Recherches sur le traitement de la phthisie pulmonaire. Paris, 1874.—Metzquer: Etude clinique de la phthisie galopante. Paris, 1874.—Fräntzel: Ueber den inneren Gebrauch von Atropinum Sulph. bei profusen Schweissen, etc. Virchow's Archiv, Bd. 58.—Weber: The Communicability of Phthisis from Husband to Wife. Trans. London Clinical Soc., 1874.—Flint: On Phthisis. Lea, Philadelphia, 1875.—Dührssen: Ueber die Behandlung der Hæmoptoe. Deutsches Archiv für klin. Med., 1875, Bd. 16, p. 93.—Friedländer: Experimentaluntersuchungen über Lungenschwindsucht and chronische Pneumonie. Virchow's Archiv, 1876, Bd. 67, p. 234.—Müller: Statistische Beiträge zur Beleuchtung der Hereditätsverhältnisse bei der Lungenschwindsucht. Inaugural Diss., Berne, 1876. Deutsch. Archiv für klin. Med., Bd. 18, p. 521.—Williams: Lettsomian Lectures on the Influence of Climate in the Treatment of Pulmonary Consumption. Brit. Med. Journ., 1876, Vol. I.—Hayden: Abstract of 330 Cases of Phthisis. Dublin Jour. Med. Sciences, 1877, II., p. 493.—Geddings: Aiken as a Health Station. Charleston Med. and Surg. Journal, 1877.—Gleitsmann: Western North Carolina as a Health Resort. Phila. Med. and Surg. Reporter, Feb., 1876. Contribution to the Treatment of Pulmonary Phthisis. New Orleans Med. and Surg. Journal, July, 1877.—McCall Anderson: Lectures on Clinical Medicine. London, 1877.—Loomis: Climatic Treatment of Phthisis. Trans. Am. Med. Assoc., 1878, p. 124.—Green: The Pathology of Pulmonary Consumption. London, 1878.—Fothergill: The Early Treatment of Phthisis. Practitioner, 1878.—Holden: Is Phthisis Contagious? Am. Jour. Med. Sci., July, 1878.—Webb: Is Phthisis Contagious? Am. Jour. Med. Sci., April, 1878.—Thompson: The Pathological Traces of Pulmonary Hemorrhage. Medico-Chirurgical Trans., LXI., p. 253.—Powell: Consumption and Certain Diseases of the Lung and Pleura. London, 1878.—Blau: Die käsige Pneumonie und Diabetes. Schmidt's Jahrbücher, 1878, II., p. 76.—Lente: The Constituents of Climate with Special Reference to Florida. Richmond and Louisville Med. Jour., August, 1878.—Chaillé: The American Mountain Sanitarium for Consumption, at Asheville, N. C. New Orleans Med. and Surg. Jour., April, 1878.—Loomis: The Adirondack Region in the Treatment of Phthisis. Trans. N. Y. Med. Society, 1879, p. 403.—Flint: Self-limitation in Cases of Phthisis. Archives of Med. (N. Y.), June and Oct., 1879.—Leaming: A New Classification of Phthisis. Archives of Med., June, 1879.—Wernich: Die Lungenphthise. Wann steckt es an? Berliner Wochenschrift, 1879, pp. 354 and 370.—Charcot: The Pathological Anatomy of Phthisis. Lancet, 1879, II., 963 and 1880, I., 182.—Brunton: Pathology of Night-sweats and Mode of Action of Strychnia, etc. St. Bartholomew's Hospital Reports, 1879.—Peter: Des temperatures locales dans la phthisie pulmonaire. Bull. de l'Acad. de méd., 2me Série, VII., No. 37. Revue des Sciences Médicales, 1879, XIV., 151.—Lebert: Traité clinique et pratique de la phthisie pulmonaire et des maladies tuberculeuses des divers organes (posthumous work), Paris 1879.—DOBELL: Loss of Weight, Blood-spitting, and Lung Disease. Philadelphia, 1879.—FONSSAGRIVES: Thérapeutique de la phthisie pulmonaire basée sur les indications, II. ed., Paris, 1880.—DENISON: Rocky Mountain Health Resorts (with Copious References to Climatic Literature), Boston, 1880.—McCALL ANDERSON: Further Remarks on the Curability of Galloping Consumption. Glasgow Journal, 1880.—HAMILTON: On Catarrhal Pneumonia and Tubercle in the Human Lung. Practitioner, 1880.—THOMSON: On Phthisis and the Supposed Influence of Climate. Melbourne, 1879. Review in Dublin Journal, May, 1880.—PETER: Leçons de clinique médicale. Tome II., Paris, 1879. Rev. in Edinburgh Jour., April, 1880. See also Schmidt's Jahrbücher, the Jahresberichte von Virchow and Hirsch, the Index Medicus, Reports of Colorado State Board of Health, etc., etc., for articles of minor importance not included in the above Bibliography.

For the sake of clearness and convenience, it is proposed to treat under this head of phthisis from a clinical point of view chiefly, but little attempt being made to distinguish the tubercular from the inflammatory form. For references and remarks as to the artifical production of phthisis and allied diseases, the reader is referred to the article on tuberculosis.

The question as to the communicability and the means of communication of phthisis from one individual to another has been brought into renewed prominence recently. In evidence of the view that the disease is not so communicated, *Cotton* brings forward the fact that out of the large number of clinical assistants and attendants connected with the Brompton Consumptive Hospital between the years 1846 and 1867, very few indeed have ever contracted the malady. The chaplain has been at the institution seventeen, the matron sixteen years. This fact speaks well for the hygienic arrangement of the hospital, and the care with which the nurses are selected, but nothing more. As *Webb* very justly observes, there is a great difference between the nursing of the phthisical in hospitals and in private practice. In the former, there is one skilled nurse to a dozen patients or so, and the nurse occupies her own apartments after being on duty for a portion of the twenty-four hours, more or less careful precautions being observed as to ventilation and the like. In the latter, the nurse is generally some member of the family or a friend, unskilled, and the circumstances under which the office of nursing is performed are apt to be such as to increase the risk of contagion.

Walshe in 1860 was not prepared to admit that the evidence of contagion was by any means conclusive, but said in 1871, "My belief in the reality of such transmissibility has of late years strengthened. I have now met with so many examples of the kind that *coincidence* becomes itself an explanation difficult of acceptance." The evidence adduced by *Weber*, *Webb*, *Holden*, *Wernich*, and others is overwhelming. *Weber* has collected the histories of twenty-nine marriages of phthisical women to healthy men, and fifty-one marriages of healthy women to phthisical men. But one of the healthy men contracted the disease, while eighteen of the healthy women fell victims. *Weber* thinks that these facts indicate transmission through the semen, but this explanation seems rather far-fetched. It is very rarely that the male is so constantly exposed to the emanations from a consumptive partner as is the female when the conditions are reversed; and the reasons are obvious. One of *Weber's* phthisical males sacrificed four wives, another three, and four two each. *Holden* thinks that the Brompton experience negatives the possibility of infection through the atmosphere where ventilation is observed, and believes "that consumption is communicable in its later stages by means of soluble excrementitious matter thrown off by the skin and deposited on the bedding or underclothing, or in any other manner brought into contact with the naked surface of a healthy body; and that, although in some instances this may be thrown off without development into new disease, it is yet very liable to be so developed, and more liable when the healthy person is by heredity or depression in a favorable state for its reception." The proviso, *where ventilation is observed*, is a very important one, especially in connection with the experiments of *Tappeiner, Schweninger,* and *Lippl,* and the clinical observations of *Reich* (see Tuberculosis).

According to the most trustworthy authorities, hereditary predisposition to phthisis can be traced only in twenty-five to thirty-five per cent of all cases of the disease, and it would seem not improbable that contagion is responsible for a share in the large proportion which remains. *MacCormac* is a strong opponent of the influence of contagion, asserting that the disease is no more communicated thus than is a fractured limb. But he holds that propagation takes place through pre-breathed air, and thus, as *Webb* shrewdly observes, inadvertently becomes one of the strongest advocates of contagion. If the exhalations from healthy lungs can produce consumption, is it not rational to suppose that such exhalations plus gases and floating particles from disintegrating lungs will have that effect in a still higher degree?

In this connection the articles of *Müller* and *Thomson* are of great interest. The former analyzes with minute care 988 cases of phthisis which came under his observation at Weissenburg, a Swiss sanitary resort. He found that heredity was traceable in thirty-five per cent of the cases from the country, while in those from cities it was so traceable in only nineteen per cent. In other words, more cases are acquired in cities, where large numbers of human beings are congregated.

The latter shows that the mortality from phthisis in Australasia has been and is steadily increasing with the increase in population, and that it is now nearly as great as in England. In fact we are learning the true relation between climate and phthisis, a disease which is eminently social. The climatic conditions are more favorable in some places than in others for the development of the disease, as will be touched on more at length under the head of treatment, but no climate can claim complete immunity from consumption.

As for contagion, the practical deductions to be drawn from a consideration of the question are plain. A physician should never sanction the sharing of the bed of a consumptive by any other person, and should enjoin upon those who must necessarily be much with such patients careful attention to ventilation, and an interval of daily exercise in the fresh air.

A recent paper of *Senator* (*Berliner Wochenschrift*, 1879, Nos. 4, 5, and 6) calls attention to the connection between phthisis and brain disease, and shows that *Ruehle's* statement (Cyclopædia, Vol. V., p. 561) of the safety with which we can infer cerebral tuberculosis whenever phthisical patients present decided brain symptoms must be somewhat modified. *Senator* reports a case admitted to hospital with phthisis of the upper portion of the right lung, the patient being up and in pretty fair general condition. Five days after entrance weakness of the right hand was noticed; this was soon followed by complete motor paralysis of the right forearm, right unilateral epileptiform convulsions, paralysis of motion in the face and right leg, high fever, aphasia, coma, and death on the twentieth day after entrance. A large abscess was found at the autopsy in the left hemisphere. The possible dependence of cerebral abscess on lung disease was first pointed out by Sir *W. Gull*, in 1858, and has since been insisted on by *Biermer*, *Huguenin*, and *R. Meyer*.

The latter collected and published, in 1867, eighty-nine cases of abscess of the brain, eleven of which were complicated by suppuration in the lungs. The pulmonary affection has often been mentioned casually, as if it could have no bearing on the abscesses, and it is to be hoped that in the future this point will be carefully investigated. *Senator* reports a second case of phthisis, in which the invasion and localization of the paralysis was very similar to that in the first, but without convulsions, aphasia, or coma; at the autopsy no localized disease could be found in the brain. In both cases the general sensibility and control of the rectal and vesical sphincters were unimpaired.

DIAGNOSIS.

Peter has made a careful study of local temperatures in phthisis and other diseases. He uses the ordinary clinical thermometer, placing the bulb on the surface of corresponding intercostal spaces on the two sides of the chest, and finds that the temperature of the affected side is always $\frac{3}{10}$–1° C. higher than that on the other. The application of this sign in doubtful cases with obscure signs is obvious. *Peter's* results are substantiated by *Vidal* (*Bulletin de l'Acad. de Méd.*, VII., No. 38).

Charteris (*Lancet*, 1876, May 13th) and *McAldowie* (*Times and Gazette*, 1878, II., 269) report on axillary temperatures in phthisis. The former found that the temperature on the affected, or more-affected, side was always higher; whereas the latter, basing his conclusions on 880 observations on 42 patients, finds that there is no law of the kind regulating the temperature. Of 359 observations in cases of unilateral and well-marked lesion, higher registrations were obtained on the healthy side 162 times, on the affected side 162 times, and 35 times the registrations were equal. Everything depends in such observations as these on minute pains and precautions, but the probability is that *Peter* is correct. *Broca's* experiments on localized cerebral temperatures are well known, and *Peter* has found local elevation in various inflammatory conditions.

The same author reports (*La France Médicale*, '78, Oct. 12th and 16th) four cases of phthisis in which venous pulsation synchronous with the systole of the heart was noted on the back of the hand. The phenomenon is attributed to paralysis of the muscular walls of the arterioles from asphyxia, and indicates that death is near at hand.

Heitler (*Wiener Wochenschrift*, 1877, Nos. 49 and 50) reports on the diagnostic value of epithelium in sputa, and denies that there are any cells the presence of which is characteristic of phthisis. All the epithelial forms which are found in this disease occur also in other pulmonary affections which do not lead to destruction of tissue.

PROGNOSIS.

Dr. *Flint* maintains the view that phthisis is, in some cases at least, a self-limited disease. He has preserved notes of and analyzed 670 cases from among those which have come under his observation in thirty-four years, the list embracing a few cases of acute tuberculosis and interstitial pneumonia, and of these 44 ended in recovery, 31 ceased to progress. Of those which recovered there was no medicinal treatment to which the arrest of the disease could be attributed in 23; and of those which ceased to progress in 15. With respect to hygienic treatment, in some cases of both groups there was no change whatever in habits of life. In other cases there were changes involving more favorable circumstances pertaining to hygiene; but a considerable portion of these changes were not of such a character that a potential influence could be attributed thereto. It is probably correct to say that the changes may have favored recovery or non-progression, but were inadequate to cause an arrest of the disease. Without wishing in any way to decry medicinal and climatic treatment, *Flint* expresses his conviction that some of the cases, the cure of which has been attributed to these measures, really got well by self-limitation. He briefly relates an instance from his own practice of recovery in New York City, without any important medication, the cavity gradually disappearing and leaving a permanent, circumscribed depression of the chest wall to mark its site.

The writer has a patient whose case is interesting in two respects. A young man, of vigorous New England stock, married soon after twenty a lady with commencing phthisis, took her to Europe and up the Nile, and was most assiduous in his devotions to her till she died of consumption, about a year after her marriage. Very soon after he began to cough, hemoptysis appeared and recurred frequently, cough and expectoration persisted. He did not care much whether he recovered or not, was reckless at times, but made several changes of climate, passing a large part of one winter in *North Germany*, and after various ups and downs during a period of six or seven years, finally recovered completely and is now a very hard-working professional man. A sufficiently long period has now elapsed—at least five years—since the cessation of all symptoms to justify the use of the term recovery. He undoubtedly contracted the disease from his devotion to his wife, but digestion remained good throughout, loss of weight, though decided, was never excessive, and I am satisfied that self-limitation played a larger part in the production of the favorable result than whiskey with cod-liver oil and change of climate.

It is, of course, an excessively difficult matter to estimate in any given case beforehand how far we can rely on this element of self-limitation, but we are not entirely without a basis for drawing conclusions as to this point. Toleration of the disease affords grounds for encouragement; the chief indication of toleration being the maintenance of the normal, or nearly the normal, pulse rate and temperature, and the retention of the power of digestion and assimilation. Again, other things being equal, the smaller the amount and extent of the lesions the better the chance of recovery through self-limitation. *Flint* states that he has been led to believe that phthisis not very infrequently ends by self-limitation before it has made sufficient progress to develop well-marked physical signs; in other words, that there are abortive cases of this as well as of other diseases, and remarks on the frequency with which the traces of small, old phthisical affection are found in bodies of those dead with various diseases.

When there are cavities resulting from the involvement of a considerable portion of pulmonary tissue, the absence of signs denoting progressive extension or general diffusion is the most reliable of the points on which to found a relatively hopeful prognosis. But Dr. *Flint's* whole article is deserving of thoughtful perusal.

Prof. McCall Anderson, of Glasgow, reports three cases of entire recovery from galloping consumption or acute tuberculosis, according to his diagnosis. The treatment was antipyretic and highly supporting, and the cases are very interesting and suggestive, even if one is not fully prepared to accept the diagnosis without question. No attempt is made in the published report to eliminate typhoid fever, whatever may have been done at the bed-side, and *Anderson* is led to found his belief in the curability of tuberculosis in general largely on a case of *Spencer Wells* (Diseases of the Ovaries, London, 1872, p. 135). A lady of twenty-two had abdominal enlargement which it was supposed might be due to a thin, non-adherent, unilocular ovarian cyst, and a small incision was made below the umbiliicus. "A large quantity of opalescent fluid escaped, and then the whole peritoneum was seen to be studded with myriads of tubercles. Some coils of small intestine were floating, but the great mass was bound down with the colon and omentum, all nodulated by tubercle, towards the free and upper part of the abdomen. The uterus and ovaries were felt to be of normal size, but their peritoneal coat was very rough." The patient made a good recovery and has since married. Now the writer inclines strongly to the belief that limited tubercle certainly, and extensive perhaps, is not so necessarily fatal as has been generally supposed, but *Mr. Wells* is not a pathologist, and every pathologist knows that small bodies are occasionally found in great numbers in the peritoneum and elsewhere which are not tubercles, but can be distinguished from them only by very careful examination—much more careful than is possible after a small opening has been made in the abdomen of a living human being, and it has been ascertained that the condition present is not that which was sought for.

TREATMENT.

It is proposed to refer first to the chief recent contributions to our knowledge as to the alleviation of the prominent symptoms of the disease and then to the treatment in general.

HÆMOPTYSIS.—The subcutaneous injection of ergotine suspended in water with or without glycerine has come more into vogue, and certainly seems to have a marked influence in some cases. It may be given in doses of five to ten grains, and repeated as often as seems necessary. Pain at the point of injection is often sufficiently prominent, but abscess is not apt to occur, and both pain and reaction are said by *Kobert* (*Schmidt's Jahrb.*, 1879, III., 241) to be much less than when sclerotinic acid is thus injected. He has also found the latter decidedly less efficacious in checking the bleeding, though *Nikitin* (*Schmidt*, 1879, I., 19) seems to have obtained more favorable results.

Pasley (*Brit. Med. Journ.*, 1880, I., 53) reports that in Trinidad at least eighty per cent of hospital cases of phthisis originate in catarrhal pneumonia and prove fatal in six to twelve weeks. At the lowest estimate, ten per cent of the cases which die in hospital terminate in profuse hæmoptysis, the patient expiring in the very act of bringing up blood.

Williamson (*Lancet*, Sept. 2d, '76) publishes his observations of one hundred and twenty cases of hæmoptysis with reference to barometric pressure and the theory that active hemorrhage takes place with increased, passive with diminished pressure. He does not find the theory borne out.

DIARRHŒA.—*Frohnmüller* (*Schmidt*, 1879, II., 230), among others, reports on the use of coto bark and its derivatives—cotoin and paracotoin—in diarrhœa from various causes, including that incident to phthisis. During the last six years he has administered the drug in ninety-two cases of diarrhœa, mostly of a colliquative character, in the course of typhoid fever and phthisis, other remedies having generally been tried without success. The preparation most commonly used was the tincture of the bark. In fifty cases the diarrhœa was checked, in twenty-six it was diminished, and in nine the drug failed altogether. As a rule, the looseness reappeared after several days and again yielded to the remedy, but in some cases no repetition was required. The drug is well borne and the appetite usually improves under its use. The writer has ordered the fluid extract prepared by Metcalf, of Boston, in some half a dozen cases without obtaining any apparent effect on the condition.

NIGHT-SWEATS.—*Lauder Brunton* (*Bartholomew's Hosp. Reports*, 1879) contributed an interesting paper on the pathology and treatment of this symptom. He thinks that sweating in phthisis is due to exhaustion of the respiratory centre by the reflex irritation from the lung, constant cough, and consequent accumulation of carbonic acid in the blood, which stimulates the sweating centres. The venosity of the blood and imperfect tissue change, not, as was formerly supposed, the mere loss of fluid, are the causes of prostration observed after night-sweats. It thus occurred to him that simply by stimulating the respiratory centre the symptom might be prevented from appearing, and putting his theory into practice he administered strychnia and nux vomica to a number of patients. The night-sweats were checked, though the drug seemed to lose its effect after a time, and, in one case, a very troublesome cough appeared to be aggravated. In cases where cough is prominent, atropia is better, this drug acting not only on the peripheral terminations of the sweat nerves, but also lessening the irritability of the sensory nerves in the lung and stimulating the respiratory centre.

Dover's powder, by diminishing the irritation from cough, tends to prevent the exhaustion of the respiratory centre, which is also powerfully stimulated by the ipecac. The favorable action of picrotoxine, reported on by *Murrell* (*Brit. Med. Journ.*, Jan. 17th, 1880), is also probably due to its stimulating qualities. Picrotoxine is the active principle of Cocculus indicus, a plant first known in Europe as a poison for taking fish, which it first throws into violent, irregular motion, and then stupefies. It had fallen into disuse as a medicinal agent till very recently. *Murrell* employs a solution in water, 1 part to 240, giving one to four minims thrice daily, the last dose at bed-time or just before the time at which the perspiration usually commences. The drug failed in only one case in which it was tried, is best given alone, and does not parch the skin, as atropia is liable to do.

There is no doubt that night-sweats are sometimes due in part to the stimulus of increased temperature on the sweat nerves, and in such cases quinine is indicated. Oxide of zinc and sulphate of copper act as astringents.

Fothergill attaches a far more direct weakening effect to night-sweats than does *Brunton* and employs largely a pill containing one-fourth of a grain of the hydrochlorate of morphia, a fortieth of a grain of atropia, with a grain of capsicum and three of aloes and myrrh. He has never seen this combination followed by any toxic effects of atropia, which is sometimes given in doses as high as a twenty-fifth of a grain. The practitioner must not go away with the impression that atropia has failed in any case until he has pushed the dose to a decided dryness of the throat and impairment of vision, flinging aside any effect upon the pupil as a fallacious test not to be trusted. Dr. *Sayre*, of New York, reports that an irregular practitioner gained a great reputation in that city many years ago in the treatment of phthisis by sponging the patient with hot vingegar containing a considerable quantity of powdered capsicum. He was very successful in arresting night-sweats, and when these are checked the appetite returns, and food is relished and digested. *Fothergill* has found these spongings useful in obstinate cases.

COUGH.—The last-named author is no friend of sedative cough mixtures or of chloral, and recommends as a pleasant and effective remedy for cough, which is at once harassing and useless, hydrobromic acid with spirits of chloroform three or four times a day. His article is a very valuable one; and it may be observed in general that German literature is not the place to which one turns for useful hints in therapeutics.

Daremberg (*Lyon Médicale*, Nov. 12th, '76) advocates the administration of creasote from *beech-tar* for the purpose of diminishing the expectoration, which is sometimes a serious drain on the patient. The purity of the remedy is of the utmost importance, and creasote from beech-tar may be distinguished from that derived from coal-tar by its behavior with collodion. Fifteen parts of coal-tar creasote and ten of collodion give a gelatinous mass when mixed, while beech-tar creasote gives a clear solution. The remedy may be administered in doses of one to three grains.

DYSPNŒA.—For remarks on the use of quebracho as a remedy for dyspnœa the reader is referred to the article on emphysema.

TAPPING A CAVITY.—*Williams* (*Brit. Med. Journ.*, 1878, I., 101) reports the case of a man of twenty-eight with a phthisical cavity in the right lung, which was tapped with a moderately large trocar, about two litres of offensive pus being evacuated; the expectoration immediately diminished in quantity and lost its fetid character, the pulse and temperature fell, and the general condition of the patient was greatly improved. The cavity was washed out several times with disinfectants.

GENERAL TREATMENT.

I. MEDICINAL.—Dr. *Andrew H. Smith*, chairman of the Committee on Restoratives of the New York Therapeutical Society, reports (*New York Med. Journ.*, April 20th, 1879) on the use of ether with cod-liver oil in ninety-four cases, as suggested by Dr. *Foster*, of London. The conclusions of the committee are as follows:

1. The addition of ether to cod-liver oil in about the proportion of fifteen minims to each half-ounce (or an equivalent amount of the compound spirit of ether) will succeed, in the vast majority of cases, in enabling the patient to take the oil, even though it previously disagreed.
2. In some cases in which the oil still disagrees after the addition of the ether, the difficulty may be overcome by giving the ether separately, from fifteen minutes to half an hour after the oil is taken.

No facts have been laid before the Committee having a bearing on the question as to whether the etherized oil is superior to the plain oil in its ultimate effect on nutrition, supposing them to be equally well tolerated by the stomach.

The same Committee reported on defibrinated blood for rectal alimentation, and, though this treatment can scarcely be classed as "medicinal," a brief notice comes in here better than elsewhere. Thirty-eight out of the sixty-three cases were of phthisis in every stage, some with large cavities, and several within a few days of death. Eight could not tolerate the injections, either because of irritability of the rectum or on account of severe and persistent colic, even when the dose was reduced to two ounces and laudanum was added. This leaves thirty cases in which the treatment had a more or less thorough trial, and of these ten showed no effect which could fairly be attributed to the injections. Some were improving before the treatment was begun, and continued improving at about the same rate during its continuance. Others were losing ground, and their downward tendency seemed not to be checked in any appreciable degree. These cases were, therefore, regarded as not affected either way by the treatment. In the other twenty cases, positive benefit seemed to have attended the use of the blood; the improvement being in some cases slight, in others very decided.

The conclusions of the committee are as follows:

1. That defibrinated blood is admirably adapted for use for rectal alimentation.

2. That in doses of two to six ounces it is usually retained without any inconvenience, and is frequently so completely absorbed that very little trace of it can be discovered in the dejections.

3. That, administered in this way once or twice a day, it produces in about one-third of the cases for the first few days more or less constipation.

4. That in a small proportion of cases the constipation persists, and even becomes more decided the longer the enemata are continued.

5. That in a very small proportion of cases irritability of the bowels attends its protracted use.

6. That it is a valuable aid to the stomach whenever the latter is inadequate to a complete nutrition of the system.

7. That its use is indicated in all cases not involving the large intestine, and requiring a tonic influence which cannot be readily attained by remedies employed in the usual way.

8. That in favorable cases it is capable of giving an impulse to nutrition which is rarely, if ever, obtained from the employment of other remedies.

9. That its use is wholly unattended with danger.

Schnitzler (*Wiener Presse*, 1876, No. 32) has employed subcutaneous injections of carbolic acid in phthisis, being encouraged to do so by success in a severe case of diphtheria which had resisted other remedies. During June and July, he treated more than one hundred cases, injecting one or two syringefuls of a one to two per cent solution once daily—though in a few cases twice—into the back or front of the chest. The result, in a large majority of cases, was diminution of the fever, marked improvement in the general condition, and in a few cases alleviation of the cough and expectoration. The injections were kept up for several weeks, and no injurious effect was observed. The patients experienced no more pain than from morphia injections, though the local burning sometimes continued a little longer. Slight, but never serious, inflammation was occasionally observed.

Experiment having indicated that phthisis may be an infectious disease, the idea was naturally suggested that an antiseptic treatment might produce favorable results—hence the employment of carbolic acid subcutaneously and by inhalation. The latest development of this idea has excited such interest in Germany that it is worthy of notice, although the brilliant success with which benzoate of soda inhalations were heralded to the profession does not stand the test of extended experience. In June, 1877, Prof. *Klebs* communicated to a scientific meeting in Munich the opinion, founded on experimental and microscopical observations, that tuberculosis is an infectious disease of parasitic nature; that it is induced by certain micro-organisms which invade the body and multiply in it, and that the expectation might be entertained of curing it by the employment of means which annihilate these organisms. One of these means is the inhalation of benzoate of soda solutions. *Rokitansky*, of Innsbruck, then took up the idea and employed a five-per-cent watery solution of the substance which was pulverized and inhaled, and his assistant, *Kroczak*, communicated to the *Wiener medicinische Presse* for Sept. 14th, 1879, "results exceeding all expectations," in fifteen cases so treated. Persons, so to speak, moribund upon entrance to the hospital were sent out cured apparently, after about three weeks of the treatment. The matter was taken up by the lay press, great excitement was caused, and the drug was forthwith tested in many of the German hospitals. Sufficient reports are now in to show that the inhalations are not only useless, but indeed harmful in some cases; causing nausea and vomiting and irritation of the air passages. Those who are curious on the subject are referred for details to articles in the *British Medical Journal* for Jan. 3d, 1880, and *Wiener Presse,* 1879, No. 51, and 1880, No. 1. Among the observers who report on the uselessness of the drug are *Guttmann*, *Senator*, *Fränkel*, and *Wolff*.

Klebs (*Allgemeine Wiener Centralzeitung*, 1880, Nos. 1, 9, 10 et seq.), however, does not give up the ship; but maintains now that the formation of miliary tubercles can be stopped, and those already formed made to vanish, by the internal administration of the benzoates, especially magnesium benzoate. And in infiltration of the apex of moderate standing, attended with high septic fever and preceded by hæmoptysis, under the inhalation or insufflation of about ten grammes of sodium benzoate, two or three times daily, and the injection of twenty to thirty grammes of magnesium benzoate daily, permanent defervescence, disappearance of catarrhal symptoms, and notable increase in weight have been observed.

II. Climatic Treatment.—It will clear the way if we here make a brief classification of phthisis, a term which represents a group of pathological conditions. This group consists, according to modern pathology, of three members, tubercle, pneumonic infiltration or exudation, and interstitial growth. It is true that in the majority of cases we find two or all three of the members of the group co-existent and intimately associated with each other; but we do, on the other hand, meet with cases in which some one of the members is alone, or almost alone represented, as in acute miliary tuberculosis, acute inflammatory phthisis, and cirrhosis of the lung—fibrous or fibroid phthisis. We are not infrequently able to determine clinically which member of the group is present or predominant in a given case, and the attempt should always be made, inasmuch as the decision has a most important bearing on the advisability of change of climate.

"Within the past few years, in this country and in foreign lands, monographs have been published with carefully prepared tables in regard to the temperature range of different health-resorts, the amount of rainfall, the degree of atmospheric pressure, the prevailing winds, the altitude, etc. Some localities are mentioned as especially desirable for phthisical invalids on account of their equability of temperature, other places are recommended on account of their luxuriant vegetation or the peculiarity of their soil. Some are thought desirable on account of their dryness of atmosphere, others on account of the humidity of their atmosphere."

"Vague and uncertain are the statements in the literature of the subject, and widely different conclusions have been arrived at by various observers. Places which at one time were the favorite resorts of consumptives have been abandoned as unhealthful and dangerous. Directly opposite views are held in regard to the therapeutic value of the same resort. An educated physician, who was in the last stage of the disease, and who had vainly tried all climates, expressed what I mean when he said to me: 'In attempting to follow the instructions of my New York medical adviser, and also those of my Philadelphia medical adviser, the one recommending a cold, the other a warm climate, I have made the result a failure.'"

"Fifteen years ago, the belief prevailed that the essential climatic element for the arrest or cure of phthisis was a warm, dry atmosphere. More recent observations and investigations have settled the fact that phthisis is not necessarily hastened in its development by a low temperature, and is not prevented or cured by a high temperature. Again, it has been claimed that the higher the altitude, the fewer were the cases of phthisis, until at a certain elevation it entirely disappeared, and that this diminution in the number of cases was due to diminished atmospheric pressure. More extended observation has demonstrated that the altitude at which this supposed immunity exists varies with the latitude; that the nearer the approach to the equator the higher must be the altitude to accomplish the desired result. This fact seems to prove that the development of phthisis does not depend upon atmospheric pressure, for the laws governing atmospheric pressure are ever the same at a given altitude."

"*Elevation* was also regarded as the cause of this immunity from phthisis. This theory was disproved by the fact that, whenever the inhabitants of elevated regions engaged in manufacturing pursuits which confined them in unwholesome air, phthisis was very frequently developed. Nevertheless, this theory so rapidly grew in favor that a large number of phthisical patients were sent to the mountains; these more markedly improved than those who were sent to the milder regions of the southern lowlands. A new series of investigations established the fact that this immunity from phthisis was not due to altitude, but to the absence of organic matter in these high elevations. In the presence or absence of these organic substances we have a very important element of difference between the air of the lowlands and the air of the mountains. That atmospheric germs are also more abundant in cities and towns has been plainly shown. Dr. *Schreiber*, in his lecture on Climatology, states that "ozone and rain have the power of purifying the atmosphere, that is, of freeing it from organic substances; that the purifying power of ozone depends on its oxidizing power; that while oxygen requires a considerable degree of heat before it will combine with other substances, ozone will do so at an ordinary temperature. Ozone destroys the products of decomposition by chemically combining with them. The air of a locality may not necessarily be unwholesome because no ozone is present, as the ozone may already have been expended in oxidizing the organic substances in the surrounding atmosphere. Yet the presence of ozone in the atmosphere is presumptive evidence that it contains no organic substances."

"The air of the ocean and high mountains is richer in ozone than that of the plains, and this substance promotes nutrition and blood changes by supplying to the respiratory organs a most active form of oxygen. Therefore, when choosing a health resort for phthisical invalids, we should give the preference to a locality in which there is constantly an excess of ozone in the air, for experience has established the fact that there the climate is especially salubrious. Experience has also shown that the direct inhalation of ozone has little if any power in preventing or arresting phthisical development, and we must, therefore, conclude that it acts simply as a purifier." (The above is quoted precisely from *Loomis:* further points will be borrowed from his paper without the use of quotation marks.) Damp warm as well as damp cold climates are unfavorable for phthisis, but the worst form of dampness is that dependent on a clayey, ill-drained soil; and this matter of soil-moisture is of the utmost importance.

Temperature has always been regarded as of very great importance in the treatment of phthisis, and a warm, sedative climate was long regarded as best for the purpose; more recently it has been claimed that a cold climate is the favorable one, and that phthisical mortality decreases as we go northward. Both views are correct to some extent. It is not the warm temperature of a locality which is so important as the absence of sudden and frequent changes, such as those to which the eastern seaboard of the greater part of this country is subject. Whether a cold or warm climate is indicated in any given case, can be determined only by the experience of the individual prior to the phthisical development. Some are greatly depressed by a cold and exhilarated by a warm climate; with others the contrary holds true. There is no evidence that temperature is directly curative.

As proof that altitude alone is too highly regarded by some, *Loomis* states that there is no place where patients do worse in all stages of the disease than among the Catskill Mountains, as he has found by experience, and he finds similar testimony given by others in regard to other mountain regions.

Sea and mountain air do not differ as widely as has been supposed by many. The latter is less dense, of lower temperature, and less humid; but they are alike in containing an excess of ozone, in freedom from organic substances and impurities, and in being cooler, and less subject to frequent variations in temperature than is the air of inland plains.

I. Individuals in whom the processes of tissue change do not require hastening are better in the mountains than on or by the sea.

II. Persons past middle life in whom phthisis has been developed do better in sea than in mountain air.

III. Phthisical invalids should not go to the mountains unless they are capable of considerable muscular activity.

IV. As a rule, phthisical subjects with an exhausted nervous system, with an overtaxed brain from excessive mental labor, or an all-absorbing occupation, yet who still retain considerable latent muscular power, will improve in the mountains, while those whose processes of tissue change require hastening or stimulating, they being in too feeble a condition to take active muscular exercise, should go to the sea.

The above conclusions are drawn under the provisional acceptance of the correctness of *Beneke's* experiments, which go to show that tissue changes take place more rapidly on or by the sea than in the mountains.

Loomis' conclusions from his own experience are as follows:

1. We can expect permanent improvement in cases of developed phthisis only after prolonged residence in the locality which experience has proved best suited to each individual case. Permanent favorable results cannot be obtained from an annual change of climate.

2. Cases of *tubercular* phthisis, in any stage of the disease, grow steadily and rapidly worse in all localities. Such cases do best in the quiet, well ventilated apartments of their own homes, where they can be surrounded by all those influences and circumstances which tend to make a feeble invalid comfortable.

3. Cases of *fibrous phthisis* in every stage, whether the fibrous process commenced in the pleura or bronchial tubes, even after retraction of the chest-walls, especially in the infra-clavicular region, is well marked, and the bronchial dilatations which accompany it give the physical signs of extensive cavities, improve and often reach a condition of comparative health when they take up their residence in regions having very high altitude, such as are found in Colorado and the Rocky Mountain ranges, where patients with asthma and emphysema derive very marked benefit.

4. Experience has led me to be very cautious in recommending these regions of high altitude to invalids with catarrhal phthisis. In the advanced stage of this form, I have never seen good results from a residence in such regions, and it is quite doubtful whether any one in the first stage has received benefit.

C. Theodore Williams (*Lancet*, Aug. 16th, 1879) says that the influence of high altitudes is intensely stimulating to respiration, circulation, and digestion; the cases in which it is indicated being those without pyrexia and with limited consolidation at one apex, limited cavity, and hemorrhagic phthisis. He is speaking largely with reference to Davos am Platz in the Swiss Alps, 5,200 feet above the sea, and so situated in a valley that the sunshine is unobstructed, while there is great protection from cold winds. The season at Davos extends from November to March, and during that time the average temperature is 23.5° F. above zero. The air is, however, so dry that patients can pass much time out of doors, and they are encouraged to sit in the fresh air and exercise as much as possible. Four hundred invalids are said to have sought this resort in 1874, from all parts of Europe, and its reputation has increased steadily since then. *Clifford Albutt* gives very favorable reports of the influence of the place on suitable cases in the *Lancet* (1877, II., Oct. 20th and 27th, 1878, I., 824).

Loomis has seen only a very limited number of cases of catarrhal (this class is apparently the same as that called by other authorities 'pneumonic') phthisis improved permanently by long sea voyages or residence in a warm climate. A large number in the early stage of the disease, going from a northern to a southern winter, are temporarily improved; after the first apparently beneficial effects are passed, the degenerative inflammatory processes go on more rapidly than before. The invalids whom he has found most markedly benefited by a sojourn during the winter months in a southern climate are those convalescing from some acute pulmonary affection, in whom the delayed convalescence raises the fear of possible phthisical development, and those in whom acquired or hereditary phthisical tendencies exist, yet in whom there may be no positive physical signs of disease of the lungs. For such cases, Aiken, S. C., Palatka, Enterprise, and Gainesville, in Florida, and Thomasville, in Georgia, are of great service.

In cases of consolidation, his best results have been obtained in those who have made a prolonged stay—one to three years—in regions of a medium elevation, 1,500 to 2,000 feet, such as Asheville, North Carolina, and the Adirondack region; a number of cases are cited by the author in illustration of the benefits derived from the latter region, and he strongly urges the further establishment of sanitariums under the charge of well educated, careful, and intelligent physicians.

Now for a few words as to some of the leading American resorts for consumptives. The climate of Florida is moist and warm, the changes in temperature between day and night being often marked, and fires being frequently needed in the evening. At Jacksonville, Palatka, Magnolia, and some other places, many comforts are attainable, and an improvement is taking place in this respect every year. With the development of railroads and the growing use of Pullman cars, the region is becoming easier of access for those who wish to avoid a sea voyage. Patients can also work gradually northward in the spring, when the heat in Florida begins to be excessive and debilitating. Thomasville is in southwestern Georgia, not elevated, but with a porous sandy soil, and large pine forests in the vicinity. It was, I believe, discovered, so to speak, by Prof. *Metcalfe*, of New York, and has one or more good hotels. Aiken, South Carolina, is also low, with a dry, bracing atmosphere and sandy soil; it is the winter home of Dr. *Geddings*. At Asheville, North Carolina, Dr. *Gleitsmann* has established a home for consumptives, at an elevation of 2,250 feet, the sunny hills of this region offering facilities for outdoor life and active exercise. In East Tennessee is Walden's Ridge ("A people without consumption," Dr. *Wight*, Chattanooga), part of the Cumberland table-land, at an elevation of 2,000 feet, but there is no provision for feeble invalids as yet in the region. The Adirondacks are too well known in this part of the country to require further mention here. In parts of Minnesota we have a dry, cold climate with medium elevation, while in San Diego and Santa Barbara, California, and Nassau, in the Bahamas, a moist sea atmosphere and very great equability of temperature are combined. Within a few years, Colorado and the foot-hills of the Rocky Mountains have attracted great attention, hough they will doubtless attract still more as the life becomes less ough, and the region better supplied with those luxuries which to many f the inhabitants of the older States have become necessities. Dr. *Denison*, of Denver, gives full details as to the benefits of this region in is recent book.

In recommending any particular health resort, careful attention should lways be given to the personal peculiarities and preferences of the patient. t is not only important that the atmosphere should be pure and the limatic conditions such that the utmost possible amount of time should e passed in the fresh air, but due regard must also be had to the opporunities offered by the resort under consideration toward the mental ccupation and amusement of each patient. There is, as a rule, but ittle use in sending the patient who abhors the simplicity of country ife to a quiet village, and for this reason, during many years to come, e shall continue to send certain patients to Europe; the greater luxury f a certain kind, novelty, and distractions of which certainly more than ounterbalance its inferiority from a strictly climatic point of view. In hort, the mind of the patient as well as his lungs must receive our arnest consideration, and a step which often involves uprooting a whole family is not one to be entered on lightly.

Dr. *Harry Leach* (*Practitioner*, 1878) writes of his personal experience of South Africa, and the reckless way in which English practitioners have sent patients thither without knowing anything about the special ocalities recommended, the means of reaching them, with the necessary expense involved, and their accommodations and special resources. South Africa is not the only resort to which his remarks are applicable, nor are English physicians the only ones who have sinned in the way he describes.

The congregation of large numbers of invalids at health resorts also exercises a most depressing influence on some people, and it would not be a bad thing if it were part of the education of physicians to spend three months in one of these places, as it was the fortune of the writer to do in Nassau, a number of years ago. The less many patients think about their own symptoms and cases the better. There is in this country opportunity for the fulfilment of every climatic indication, and we have entered on a great era of progress in the "home treatment" of consumption.

TUBERCULOSIS.

BIBLIOGRAPHY.

Friedländer: Ueber lokale Tuberculose. Volkmann's Sammlung der klinischen Vorträge, No. 64, 1873.—**Wolff**: Entzündliche Veränderungen, etc. Virchow's Archiv, Bd. 67, 1876, 234.—**Metzquer**: Archives générales, XXX., 1877, 239.—**Malmberg**: Zur Geschichte der Inokulationstuberculose, etc. Hygiea, August, 1877.—**Foot**: Two Lectures on Tuberculosis. Dublin Journal, II., 1877, pp. 1 and 101.—**Reich**: Die Tuberculose, eine Infektionskrankheit. Berliner Wochenschrift, 1878, p. 551.—**Tappeiner**: Eine neue Methode die Tuberculose zu erzeugen. Virchow's Archiv, 1878, Bd. 74, 393.—**Schottelius**: Experimentelle Untersuchungen über die Wirkung inhalirter Substanzen. Archiv f. path. Anat. und Phys., LXXIII., p. 524.—**Lippl**: Allgemeine med. Central-Zeitung, 1878, XXXVIII., 461.—**Ziegler**: Tuberculose. Volkmann's Sammlung, 1878, No. 151.—**Spillmann**: De la tuberculisation du tube digestif, Paris, 1878 (with full bibliography of the subject).—**Schüller**: Therapeutische Versuche bei tuberculösen Thieren, etc. Arch. für exp. Pathologie, Bd. XI., p. 84.—**Epstein**: Tuberculose im Säuglingsalter. Prager Vierteljahrschrift, 1879, 103.—**Orth**: Experimentelle Untersuchungen über Fütterungstuberculose. Virchow's Archiv, 1879, Bd. 76, p. 217.—**Weigert**: Zur Lehre von der Tuberculose und den verwandten Erkrankungen. Virchow's Archiv, 1879, Bd. 77, 269.—**Bollinger**: Künstliche Tuberculose erzeugt durch den Genuss der Milch tuberculöser Kühe. Aerztliches Intelligenzblatt, 47, 1879. Allg. med. Centralzeit., 96, 1879.—**Aufrecht**: Zur Kenntniss der Scrophulose und Tuberculose. Deutsch. med. Wochen., 1879, No. 50. Allg. med. Centralzeit., Dec., 1879.—**Virchow**: Ueber die Perlsucht der Hausthiere und deren Uebertragung durch die Nahrung. Berliner Wochen., 1880, Nos. 14 and 15.—**Cohnheim**: Die Tuberculose vom Standpunkte der Infektionslehre. Leipzig, 1880.

The nature of tuberculosis and its relations to scrofula and phthisis are still questions which engage some of the best minds in the profession, and towards the ultimate solution of which important contributions have been made during the last six years, though its solution may be, and probably is, still far off. At the time of the publication of the second edition of the Encyclopædia, the giant cell was regarded by many as the characteristic anatomical element of tubercle and tubercular products, but it has more recently been shown that these cells are found in other and widely different formations. In fact, suffice it to say, we are acquainted to-day with no anatomical criterion of any kind which stamps a formation as indisputably tubercular. From a theoretical point of view, this admission is very discouraging and unsatisfactory, but the practical results are not so serious as might be supposed, inasmuch as any reasonable doubt can be removed, in the great majority of cases, by consideration of the circumstances and origin of the formation, its localization, distribution, symptoms, and termination.

Aufrecht says that miliary tubercle is not the anatomical basis of tuberculosis, a term which has merely an etiologico-clinical meaning, the granulation being the result of various causes, and never the primary change in that condition which has so long been known under the name tuberculosis. In perfect analogy with the term scrofulosis, the term tuberculosis means simply this—a tendency, generally hereditary, to weakness of certain organs, in consequence of which weakness they become readily the seat of inflammatory processes and resulting cheesy degeneration of the tissue. Pulmonary consumption is the most common localization and manifestation of tuberculosis. Scrofula and tuberculosis are brothers, as they generally appear in the same families. If some members of a large family have died of scrofulous affections, some of the survivors are pretty sure to have tuberculosis later in life. Moreover, individuals who in childhood suffered from the former, are more likely than other people to develop the latter as they grow older. Finally, tuberculosis in the parent is eminently a cause of scrofula, and eventually tuberculosis, in the children. This view is directly opposed to that of those who believe tuberculosis to be an infectious disease like syphilis, whether low organisms play a part in it or not. If it is an infectious disease, how can so many people recover from it, and how can we expect to combat a chronic, infectious disease by simple hygiene? This is not the way in which we cure syphilis. [It is not obvious why people should not recover from an infectious disease, and the best authorities are unanimous in insisting on the very great importance, and even curative influence, of hygiene in syphilis.]

In view of the fact that the theory of the infectious nature of tuberculosis has necessarily been based on experiments on the lower animals, great interest is attached to the observations of *Reich,* which indicate the transmission of the disease from a phthisical midwife to a number of children. The village of Neuenburg is healthily situated on the Rhine, counts 1,300 inhabitants, and gave occupation to two midwives. One of these was healthy, the other began to suffer from a pulmonary affection in the winter of '74–'75, and when examined by *Reich* in July, 1875, had several cavities in the right lung, with profuse purulent expectoration. She nevertheless pursued her occupation almost uninterruptedly up to the time of her death, July, 1876. Of ninety-two children who died during their first year in Neuenburg, between 1866 and 1874, tubercular meningitis was the cause of death in but two cases; of twelve who died in 1877 in but one case, and in that there was hereditary predisposition. Now, between July 11th, 1875, and September 29th, 1876, tubercular meningitis caused the death of *ten* children who were born between April

4th, 1875, and May 10th, 1876, of healthy parentage. All of these ten cases occurred among the clients of the consumptive midwife, and not a single case of the kind among the clients of the healthy midwife. The disease began with bronchial catarrh, which was followed by well-marked symptoms of tubercular meningitis, though in no case does an autopsy seem to have been made, and it was found that the midwife was accustomed to suck the mucus out of the mouths of new-born children under her care, to blow into their mouths if there was any asphyxia, and to subject them in general to an unusual degree of kissing and fondling.

The important bearings of the above report are too obvious to require comment, and in this connection the experiments of *Tappeiner* are of great interest. He caused, namely, dogs to inhale the spray of human phthisical sputa rubbed up and diluted with water, dogs being chosen on account of their great relative immunity from tubercular disease. Eleven animals were subjected to the treatment, some for longer, others for shorter periods, during four to eight weeks, and though only two of these showed any signs of sickness, all were found to present marked evidences of disease when they were killed. In all but one miliary tubercles were found, especially in the lungs, but often also in other organs, and that one had extensive degenerative pneumonia. The amount of sputum used seems to have been very small, and the earliest period at which the nodules appeared was during the third week of the inhalation. In order to determine whether the transmission of the disease took place really through the air-passages, or through the digestive tract, the particles of sputum which lodged in the mouth and pharynx being swallowed, *Tappeiner* fed some dogs with portions of the same sputum which was used in the other experiments. He thus produced miliary tubercles, but not with the same constancy as under the other method. *Schottelius* was led by these experiments to see what effect follows the inhalation of powdered cinnabar, brain, cheese, and bronchitic sputum, likewise rubbed up and diluted with water. Miliary nodules were found in the lungs in all cases, in equal quantity with both phthisical and bronchitic sputum. Cheese produced a smaller quantity, brain still less, and cinnabar least of all, namely, a few whitish nodules with pigmented centres. He consequently concludes that powdered mineral substances give rise to little irritation, but that powdered organic matter, especially sputum, sets up broncho-pneumonia, which may appear in small foci, and bear a great resemblance to miliary tubercles. Bronchitic sputum gives rise to precisely the same lesions, and the cause is not specific, but simply the irritation of an organic matter, which is in a state of fine subdivision, and prone to decay. This view is shared by *Ziegler*, who considers true miliary tubercle as the indication of a general infection. *Weigert*, however, considers the nodules produced by *Tappeiner* as genuine tubercles, while agreeing that those of *Schottelius* were simply inflammatory; and this for the reason that in the experiments by the former the nodules were found in other organs as well as the lungs, while in those of the latter they were not.

Chauveau was the first to experiment on the transmission of tuberculosis through the intestinal canal, and very recently the subject has been taken up by *Bollinger*, *Orth*, *Virchow*, and others. The two former hold that the pearl disease of cattle and human tuberculosis are one and the same disease, but the latter is more cautious, and his lecture is well worth a detailed abstract, showing as it does, not only the present state of our knowledge on the relation of these affections, but also the difficulties which are involved in investigations of the kind.

For four years *Virchow* has been experimenting in the Royal Veterinary School in Berlin, at the instance of the Minister for Agriculture, to ascertain how far the secretions and flesh of animals, especially cattle, with the pearl disease are prejudicial to man, what bad effects may be produced, and what sanitary measures should be adopted by law for their prevention. He is apparently led to make known his results thus far by the unduly positive statement of the first-named authors and *Klebs*, who thinks that he has discovered a special germ or parasite, the common cause and carrier of tubercle and the pearl disease.

In the first place, confusion has been introduced into the question by those who fail to recognize due distinction between the characteristic products of the pearl disease and those of chronic peribronchitis and pneumonia. Both affections are common in cattle, and are sometimes co-existent but their products differ greatly as regards their life-history. The pearl disease is characterized, namely, by the presence of hard tumors which increase in size with age, scarcely ever become cheesy, and consequently do not lead to ulceration, but have a marked tendency to cretification. These are important points of distinction, and in the present state of our knowledge we shall do well not to interchange the terms pearl nodules, inflammatory products, and tubercles.

It is a cause of difficulty that our common domestic animals are not well suited to experiment on the subject, all being subject to diseases which are more or less similar to the tubercular, or may induce tuberculosis itself. Dogs are completely exempt from tuberculosis by ingestion —they can eat what they choose; and in rabbits, guinea-pigs, cats, pigs, goats, and sheep it is first necessary to study the exact pathology of the nodules and cheesy or calcified masses to which they are subject. Echinococcous disease is very common in pigs' livers, but here we also frequently meet with nodules which, when they first appear, are no larger than miliary tubercles, but increase in size without ever becoming cheesy, and have a great resemblance to those of cancer. Time will enable us to distinguish between these various conditions and tuberculosis, but then a new difficulty arises: it is, namely, not to be directly concluded that the changes found are the result of our experiments. Suppose we take a young and apparently healthy pig, and feed him daily for five or six weeks with pearl-disease products, the question arises whether the various changes are due to the food. From extreme antiquity the pig has been notoriously subject to swellings of the lymphatic glands, especially the submaxillary and jugular; the very term scrofula has this origin, and signifies a condition in man similar to that which occurs in pigs. These glands are by no means easy to feel, far less the mesenteric and bronchial glands, and it may well happen that we pronounce a pig healthy when, in fact, his glands are more or less enlarged.

Again, not a single experimenter in this field pretends that his results are constant and invariable. Analogous changes are found in animals which have not been fed with pearl-disease products; as to the frequency of these changes, we have no statistics at our disposal, but *Virchow's* experience satisfies him that they are certainly common in the pig and coincide in their course with what are called scrofulous glands in the human subject. The process begins with cellular hyperplasia, which then becomes cheesy and very often calcified, but is not followed by the development of nodules—tubercles. In the majority of cases, as in the human subject, the cervical glands are chiefly affected, but in some cases the mesenteric, in others the thoracic, present the most marked changes. It is not, however, unusual for one of these three groups to be alone affected, and genuine tubercular affections are very rare, provided that the name tubercle is not applied where it does not belong. Much more weight is to be attached to small nodules or tubercles within the different organs than to the glandular swellings: the more common the lesion the greater the allowance which must be made for accident.

Pregnant sows have been bought and cared for until after the birth of their young; the litters have been preserved, and when they have reached a certain size, fed with meat or milk from cattle with the pearl disease, some animals from each litter being kept under natural conditions for purposes of comparison. In some litters the submaxillary and cervical, in others the mesenteric glands were chiefly affected; but the latter not so frequently as we would naturally expect, the supposed poisonous material being introduced into the digestive tract; the intestine itself remained invariably free from changes. It is difficult to understand how the submaxillary glands should be the seat of changes under these circumstances, while the mesenteric are unaffected, and *Bollinger's* explanation is more ingenious than satisfactory. He supposes that in these cases the virus is completely absorbed in the mouth, but this is utterly inconceivable. But there is still another difficulty. If a young pig eats daily a material which is always poisonous, one would naturally expect the changes thus induced to present a constant and steadily progressive character: it is true that this does happen, but only very exceptionally. In the great majority of cases, the lesions which are found after four to six weeks' feeding are already relatively old, instead of being in various stages of development. One fact is, however, unquestionable, and all experimenters are agreed upon it. More animals, namely, become diseased among those thus fed than among those kept for comparison and leading the ordinary life of domestication.

During the series of experiments, a most instructive mistake was made. A cow was purchased, after being pronounced by the best veterinary authorities as the subject of the pearl disease, and a number of animals of various kinds were fed upon her milk. After a time the cow was killed, and no trace of the pearl disease was found, but the lung-tissue was almost entirely replaced by echinococci! The animals fed on her milk were killed before the cow; a calf which had been thus fed during a period of three months presented no recent lesions, but partially cheesy masses were found in the lungs and various glands, which latter looked exactly like those found in the subjects of the pearl disease. Others of the animals presented various changes, even some of those fed with the milk after it had been boiled, and others were unaffected. If the cow had not been killed, the results would have been considered brilliant, though it must be confessed that a larger number of animals of this series escaped disease than of another, in which the milch cow proved really to have the pearl disease.

Virchow does not feel warranted in going further at present than to suspect that pearl-disease products are injurious. The lesions of the disease are never found in the muscular tissue which is used for human food, and we are scarcely warranted as yet in taking legal measures to prevent the consumption of those parts. No human being has ever yet got a pearl tumor from eating the flesh of an animal with that disease. Some experiments have also been made with the meat of horses and beef more or less in decay, but the results do not as yet show much, one way or the other. In regard to milk, it appears probable that a distinction must be drawn between the milk of cows with local manifestations of the pearl disease in the udders, and those free from this complication. In a case of the former kind, large numbers of micrococci were found in the milk, but whether they were the peculiar cocci of this disease is not known at present.

Epstein's experience leads him to think that children at the breast acquire tuberculosis chiefly through the nurse's milk. He has seen large numbers of children of tubercular parentage, and observed that most of them did well when given to healthy nurses, and kept away from contact with tuberculous people; some died of chronic catarrhal processes in the lungs, but in no one of these instances were tubercles found at the autopsy. On the other hand, children suckled by tubercular mothers became tuberculous very soon after the appearance or the exacerbation of the pulmonary affection of the mother. In one of his cases, the tubercular changes were chiefly marked in the mesenteric glands and intestinal canal. In other words, nurslings generally ingest the disease, while older children inhale it.

Metzquer holds that the ingestion of tuberculous food is never a means of the inoculation of phthisis. After a certain time, irritation and inflammation of those parts of the intestinal canal with which the material remains longest in contact ensues, giving rise to diarrhœa and loss of weight. Hyperæmia and interference with the circulation result in intestinal thrombosis, and the inflammation may result in ulceration; in the latter event, and in that event only, is the way opened for infection.

A remarkable pamphlet has come from the pen of *Cohnheim* within a very few months, bringing forward fresh evidence that tuberculosis may be propagated by a specific process of infection, and propounding an exact and complete theory of the origin of the disease in the human subject. The original paper has not as yet come to hand, and the following summary is taken, with but few changes, from the *British Medical Journal* for May 8th, 1880, it being expressly stated that the exposition is, as far as possible, almost literally in the words of *Cohnheim* himself.

In the theory which was originated by *Virchow*, long adopted in Germany, and now largely taught in this country, it is held that a fundamental difference exists between miliary tubercle—tubercle properly so called—and the inflammatory products found in scrofulous diseases of the lymphatic glands, and in the form of phthisis called by the Germans caseous pneumonia. The latter processes are regarded by *Virchow* as purely inflammatory in their nature, and as differing from other such processes only in that there is neither absorption nor further development. This theory is based on certain anatomical facts, and may be conveniently termed the anatomical theory. One of *Cohnheim's* objects is to show that, since *Villemin* has proved that tubercle can be transmitted by inoculation, this anatomical theory has been completely shattered. The test for tubercle no longer consists in anatomical elements or arrangement of elements, but in the capacity of a morbid product, when introduced into the body of a rabbit or guinea-pig, to produce tuberculosis in the animal. What produces tuberculosis is tubercle; what fails to produce tuberculosis is not tubercle. Judged by this criterion, caseous pneumonia and miliary tubercle are manifestations of one virus, and the doctrine of *Laennec*, which for a time had been in the shade, has now received ample confirmation. Further, the scrofulous lymphatic gland is found to contain the same poison. Other simple tissues, such as that of lupus, are found not to be inoculable.

For the application of this test all animals are not equally reliable. Dogs, for example, show little susceptibility, while rabbits and guinea-pigs have it in a remarkable degree. The way in which the tuberculous substance is introduced into the animal is almost a matter of indifference; the common and most convenient method of procedure is inoculation by a small incision, either into the subcutaneous tissue, the pleural and peritoneal cavities, or the anterior chamber of the eye. Whether there is much or little tubercular substance employed, whether it is used alone or mixed with other tissues—as, for example, pieces of lung containing tuberculous nodules—is of little importance; of far greater consequence, on the other hand, is it that the substance used should be fresh and free from decomposition. The fresher it is, the less likely is the experiment to be affected by septic and similar influences, and infection is the more certain. How the infection takes place is best seen in animals when the tubercular matter has been introduced into the anterior chamber of the eye. In this case, if the substance be absolutely fresh, the irritation produced by the introduction soon passes away, the small portion introduced becomes gradually smaller and smaller, and may even completely disappear. Then, for a time, the eye is perfectly clear and intact, until suddenly a larger or smaller number of minute gray nodules appear on the iris, grow to a certain size, and then caseate, exactly as happens with human tubercle. In the rabbit *Salomonsen* and *Cohnheim* observed the eruption usually about the twenty-first day after inoculation; in guinea-pigs, as a rule, a week earlier. In the rabbit, also, the period of incubation is sometimes shortened to fourteen days.

It is this test that, applied to human tubercle on the one hand, or caseating scrofulous products on the other, shows that they are both caused by the same virus. All these morbid products are effective, and in the same degree. If a piece of tubercular peritoneum or cerebral membrane be introduced into the peritoneal cavity of a rabbit, typical tuberculosis, beginning in the abdominal organs, is the result. But inoculation with a piece of lung affected with caseating pneumonia, or of a caseating testicle, produces exactly the same effect, and nothing succeeds better than a freshly excised scrofulous lymphatic gland from the neck.

Those who are convinced that all contagious virus is due to a parasite, will, of course, not hesitate to believe that the tubercular poison is corpuscular, and that, in a not very distant future, this corpuscle will be demonstrated in tubercle and in scrofulous products. Until, however, this goal has been reached, there remains no other test for tubercle than that of infection. The leading principle here, as in all infecting maladies which produce local changes, is that a tubercular or scrofulous product is generated wherever the tubercular virus is present and remains for a certain time. The chief factor, therefore, in determining the localization of the disease, is the mode of introduction into the system. Once introduced, its further extension is influenced by the conditions of the structure of the part, and takes the direction of the natural roads of the part. Consequently, whilst, on the one hand, the course which the disease takes is very different in different cases, the eventual entrance of the virus into the circulation affords the conditions for the development of tubercle in distant organs. The influence of the point of introduction is strikingly exemplified by inoculation experiments. Where a small piece of tubercle is introduced into the peritoneal cavity, there constantly follows, in the first instance, tuberculosis of the peritoneum, liver, and spleen; after inoculation into the anterior chamber of the eye, the iris is first affected; after feeding with tubercular matter, the intestine and the mesenteric glands; after the inhalation of pulverized sputa, the lungs and bronchial glands; and, when the material has been introduced into the subcutaneous connective issue, it is the nearest lymphatic glands in which the disease first develops.

That the lungs are affected with tubercular disease in a far larger ratio than other organs shows, in Professor *Cohnheim's* opinion, that the poison is introduced into the human economy most frequently with the inspired air. This conception finds support, he believes, in the frequency with which the bronchial and tracheal lymphatic glands are affected in a very early stage of the disease—so much so, that an extensive tubercular pleurisy, and still more frequently advanced caseation of the glands, is found whilst in the lungs there are only a few nodules or a very scant amount of caseous infiltration. An analogous condition is found in the certainty and rapidity with which inhaled carbonaceous particles reach the pleura and bronchial glands. The conditions which determine whether the inhaled virus shall produce disseminated tubercles or caseous pneumonia are still unknown.

Whilst the pleura and bronchial glands become diseased immediately after or simultaneously with the lungs, the anatomical relations of the air-passages with each other and the digestive canal, furnish the means for further development of the disease. So soon as the tubercular products break down and ulceration takes place, a certain amount of tubercular, that is to say, of infecting matter, must leave the lungs. This reaches the trachea and larynx, and, if it take root there, tubercle and tubercular ulceration are the consequence. Then come the pharynx, the soft palate, the root of the tongue, and the tonsillar region, which are all exposed to the same influence. A certain proportion of the matter is swallowed, but the passage through the œsophagus is too rapid, and undoubtedly the acid reaction of the gastric juice too adverse to the local inoculation and further development of the organized tubercle poison, to favor the disease in the stomach and gullet, where tuberculosis is exceedingly rare. When, in consequence of the swallowing of large quantities of tubercular matter, catarrh of the stomach has been produced, the chief obstacle to the transmission of the virus into the intestine is overcome. In the bowel, infection will most likely follow at points where the intestinal contents are longest detained; that is to say, in the neighborhood of the ileo-cœcal valve, the lower portion of the ileum, the cœcum and ascending colon. The upper and lower extremities of the tube are less exposed. It is easily ascertainable in what parts of the intestinal wall the tubercle is most likely to fix itself—on those points, namely, where all absorbed substances are first retained; that is to say, in the lymphatic structures, the isolated and grouped lymph-follicles. These are, in fact, as is well known, the seat of caseating and tubercular ulcers of the intestines. Simultaneously with or immediately following the development of the virus in the follicles comes the development in the mesenteric glands; whilst, on the other hand, the tubercular ulcers admit the poison into the branches of the portal vein, and the liver is then endangered. How frequent tuberculosis of the liver is, every one knows who has taken the trouble to examine this organ with sufficient care in all cases of chronic tuberculosis. With this combination, consumptive disease of the lungs and intestines frequently ends, but further developments sometimes take place. The disease may enter the ductus choledochus and produce tuberculosis of the bile ducts. More frequent and more important is transmission to the peritoneum from deep ulcers of the intestines. And the virus inhaled with the atmospheric air may take root directly in the larynx and trachea, producing the so-called primary tuberculosis of the larynx.

Whilst the tubercular disease of the digestive tube which has been described in the foregoing sketch is secondary to the affection of the lungs, there are cases in which the intestinal canal admits the virus into the system for the first time. Amongst this class of cases, *Cohnheim* reckons advanced tubercular disease of the mesenteric glands and peritoneum, without disease or with only very slight disease in the lungs—a form of the affection rare in adults, but unfortunately very common in young children. The reason why young children are specially exposed to this danger may, perhaps, be found in the experiments of *Gerlach*, *Klebs*, *Orth*, and others, who have shown how close is the connection between the virus of the consumptive disease of cattle and that of human tuberculosis, and have further proved that the virus from the diseased cows is contained in their milk. Whether the milk of tubercular women contains the virus does not seem yet to have been investigated [see the observations of *Epstein* referred to above]; but, with the well-known prevalence of tuberculosis among cattle on the one hand, and with the prevalence of the habit of bringing up children on cow's milk on the other, an explanation of the frequency of primary consumptive disease of the intestines is ready to our hand. Perhaps the influence of food in producing tubercular disease has even a wider extent. The question may at least be entertained whether all the so-called scrofulous inflammations of the lips, mouth, and pharynx, and especially the caseating swellings of the lymphatic glands of the neck, are not due to the direct inoculation of the tubercular virus contained in food, and more especially here, also, in infected milk.

Cohnheim's remarks on uro-genital tuberculosis are very suggestive. Direct infection from one sex to the other, as by syphilis, although not impossible, is scarcely to be expected. If the virus from tuberculosis of the uterus ever is transmitted *in coitu* to the male urethra, or perhaps, what is not so unlikely, if a man with tuberculosis of the lung or other organ can transmit the virus to the genital mucous membrane of a woman, such cases must still be very exceptional. As a rule, uro-genital tuberculosis is a disease of excretion. The virus, from whatever source it has entered the blood, is excreted by the kidneys, and doubtless, like cinnabar particles, oil-drops, milk-globules, and bacteria, through the glomeruli. It by this means gets access to the urinary passages, and, on whatever point it gets a hold, tuberculosis develops. Most frequently, this takes place in the open canaliculi of the pyramids; but the disease may develop also in the pelvis of the kidney, in the ureter, and the bladder, and even in the prostatic portion of the urethra tuberculosis may occur. It may cross in the bladder to the other ureter, and ascend to the other kidney; more frequently, however, it is the urethra that is attacked. In the male the virus attacks the prostate, thence passes by the *ductus ejaculatorii* to the seminal vesicles, and onwards to the vas deferens, the epididymis, and testicle, but it can also pass directly where the ureter and the vas deferens cross. In the female, the anatomical arrangement of parts renders the route by the urinary passages very improbable. In the great majority of cases, the parts affected are the Fallopian tubes and the uterine mucous membrane, the virus entering the former from the peritoneum, which is scarcely ever found free in genital tuberculosis in the female.

Although analysis shows that uro-genital tuberculosis is scarcely ever the primary outbreak, this cannot be said of all the parts where tubercle is localized. In meningeal tuberculosis, there are cases, especially in children, in which the usual deposits in the lungs and glands are not to be found. How, in such cases, and even in those in which the deposits in other organs are present, is the transmission of the virus to the membranes of the brain to be explained? *Cohnheim* is in such instances disposed to accept *Weigert's* explanation, who has suggested that the poison gets access to the membranes of the brain from the nose, passing through the foramina of the ethmoid bone.

Still more difficult of explanation are cases of tubercular disease of bone and fungating tubercular disease of joints, occurring in individuals in whose other organs no tubercular deposit is to be discovered. Although in most such cases the development of the local disease is traced to an injury, yet it is not to be supposed that an injury can beget the tubercular poison. *Cohnheim* conceives it to be possible that, in such persons, the virus is already circulating in the blood, and that, with the exudation which follows the injury, the poison leaves the blood and engrafts itself in the tissues. As a rule, tubercular poison is not spread through the medium of the circulation, though in favorable instances this occurs; and metastatic tubercle, comparable to the most developed forms of metastatic inflammations and tumors, is sometimes found.

When we add to this mode of infection the direct transmission from the peritoneum to the spleen, through the lymphatics of the diaphragm to the pleura, from the pleura to the pericardium, etc., we need not wonder at the number of organs that are sometimes found affected with tuberculosis.

In explanation of the instances of acute miliary tuberculosis come the cases described by *Ponfick*, in which, as a cause of the contamination of the blood with large quantities of the poison, tuberculosis and infiltration of the thoracic duct were found. A more frequent cause, however, of this rapid form of the disease is tuberculosis of the blood-vessels of the lungs, instances of which are recorded by *Weigert*. A local tuberculosis of the pleura, bronchial glands, or mediastinum has extended into the wall of a pulmonary vein, and a large surface of caseating tubercular substance has projected into the lumen of the vessel. In the light of these discoveries, acute miliary tuberculosis loses much of its mystery.

When the chronic nature of scrofulous affection of the lymphatic glands, and the immunity to the general health that often follows it and scrofulous diseases of the joints, are compared with the rapid and deadly course of acute tuberculosis, it may seem extraordinary that they are all attributable to the same poison; but inoculation of rabbits shows that they all produce the same disease—a pure tuberculosis. In endeavoring to account for the comparative innocence of some classes of cases, it must not be forgotten that the human organism may entirely overcome the poison. Tuberculosis can heal. Pathological anatomy has long known cretification and cicatrization after tubercular deposit and ulceration. *Cohnheim*, assuming the error of the dualistic theory of syphilis, compares the localization of tubercular affections with cases of syphilitic infection in which there is no manifestation of the disease beyond the nearest lymphatic glands. Individual differences in the behavior of the organism which has been infected by tubercle are seen even in inoculated rabbits. When equally large pieces of the same caseating lymphatic gland are introduced into the peritoneal cavity and the anterior chamber of the eye of a number of guinea-pigs or rabbits, the first outbreak of tuberculosis takes place at about the same time and in the same manner in all of them, but the further progress shows the greatest imaginable diversities. One animal succumbs after five weeks, and, on examining the body, nodules and caseation are found in almost every organ—the peritoneum, liver, spleen, lymphatic glands, the lungs, vascular walls, etc. A second lives over two months; a third, three months or more; in another, the respiratory organs are almost entirely free, while the abdominal organs are extensively affected; in another, the eye is completely destroyed by caseous panophthalmitis, whilst the animal is otherwise perfectly healthy, is not emaciated, eats well, and is strong and lively; and when, finally, after many months, it is killed, tubercle is found nowhere except in the eye.

Cohnheim will not have it that the different powers possessed by individuals of resisting the tubercular poison are due to the presence or absence of a phthisical or consumptive habit of body. The so-called consumptive habit has nothing to do, he believes, with a facility for receiving the virus, but is a product of the disease. Individuals with this phthisical habit of body are not specially disposed to tubercle, they are already tubercular. In their case, the disease has been either acquired in early life or has been inherited. That tuberculosis is hereditary is too well known to require to be more than stated; but, translated into other language, that means nothing more or less than that the virus can be transmitted in the semen and in the ovum; whether it can, like the virus of small-pox, be transmitted to the fetus through the placental circulation remains yet to be learned. The only difference, in this respect, between it and the syphilitic poison is that, while the latter manifests itself in the offspring before or soon after birth, in the case of the tubercle poison, development takes place at a later period.

Thus, in the whole history of tubercle, everything is due to the special nature of the poison and its effects. According to the present manner of looking at the subject, every man is tubercular in whose body the tubercular poison has taken root. There is, accordingly, no predisposition for tubercle any more than for syphilis, although some persons are more easily infected than others. Nor is there any constitutional predisposition that causes hyperplastic or inflammatory products to caseate like tubercle instead of being absorbed or changed into connective tissue, but only those pathological products undergo the specific change which are already the product of the tubercular virus. Other than this, there is nothing in the whole course of tuberculosis wherein there is a difference in principle between it and other diseases arising from local infection. Not even the fever is pathognomonic.

This brilliant generalization is to be submitted to the test of time and criticism; but the source from which it emanates commands respectful attention, as does the vast practical and theoretical importance of the subject.

The whole question of tuberculosis is in such an unsettled state that the purpose of this article has been merely to indicate the drift and results of recent investigation, without attempting criticism.

DISEASES OF THE HEART.

DISEASES OF THE PERICARDIUM.

BY

T. M. ROTCH, M.D.,

OF BOSTON,

PHYSICIAN TO THE BOSTON DISPENSARY; CLINICAL INSTRUCTOR IN DISEASES OF CHILDREN IN THE
HARVARD MEDICAL SCHOOL.

HYPERTROPHY AND DILATATION OF THE HEART.

BIBLIOGRAPHY.

Ustimowitsch: Beiträge zur Theorie der Harnabsonderung. 1871, S. 198.—Gull and Sutton: Bright's Disease and Contracted Kidney. Med. Chirur. Transact., Vol. LV.—Berthiot: Grossesse et maladies du cœur. Paris, 1876.—Ménière: Grossesse et maladies du cœur. Archiv général de méd., XVI., p. 521.—Marty: Des accidents gravido-cardiaques. Paris, 1876.—Casanova: La grossesse dans ses rapports avec les maladies du cœur. Thêse de Paris, 1876.—Fritsch: Ein Nachtrag zu den Bemerkungen zur Patholog. und Physiolog. des Cirkulationsapparates bei Schwangern und Wöchnerinnen. Archiv für Gynäkol., X., 2, p. 270, 1876.—Löhlein: Ueber das Verhalten des Herzens bei Schwangern und Wöchnerinnen. Ztschr. für Geburtsh. und Frauenkrankh., I., 3, 482, 1876.—Barr: Notes on Cases of Disease of the Heart with Remarks on the Effects of Over-work. Edinburgh Med. Jour., Dec., 1876.—Brudi: Herzhyper. als Folge von ausgedehnter Pleuraverwachs. Deutsches Archiv für klinische Med., Jun., 1877.—Bäumler: Ueber Obliteration der Pleurasäcke und Verlust der Lungenelasticität als Ursache von Herzhypertroph. Deutsches Archiv für klin. Med., XIX., S., 471.—Münzinger: Das Tübingen Herz. Deutsches Archiv, 5 June, 1877.—Macdonald: The Relation between Chronic Disease of the Heart and Pregnancy. Obstet. Journ., May, Nov., 1877.—Ewald: Ueber die Veränderungen klein. Gefässe bei Morbus Brightii und die darauf bezügliche Theorie. Virchow's Archiv, Bd. LXXI., S. 466, 1877.—Zunker: Ein Fall von Dilatation und Fettmetamorph. des Herzens in Folge Ueberanstrengung durch schwere Arbeit. Berlin klin. Wochensch., Nos. 17–18, 1877.—Senator: Beziehungen der Herzhypertroph. zu Nierenleiden. Virchow's Archiv, V., 73, S. 313, 1878.—Buhl: Ueber Bright's Granularschwund der Nieren und Herzhypertroph. Mittheilung aus em pathol. Institut zu München, 1878, S. 38.—Porak: Influence de la grossesse t des couches sur l'appareil circulatoire et troubles gravido-cardiaques. Revue es Sciences médicales, V., X., 342.—Hirt: Die äusseren Krankheiten der Arbeiter. eiträge zur Förderung der öffentl. Gesundheitspfleg., II. Abtheilung, 1878.—Rawitz und Israel: Experiment. Untersuch. über den Zusammenhang zwischen ierenkrank. und Herzhypertroph.—Cohnstein: Ueber puerperal Herzhyper- ph. Virchow's Archiv, Bd. 77, S. 146.—Lewinski: Die Störung. im Circula- nsapparat, chlorotische, und die functionelle Mitralinsuffic. Virchow's chiv, Bd. 76, S. 292.—Ziemssen: Fall von spontan. Herzruptur, Hypertroph. d Schrumpfung der Nieren. Schmidt Jahr., 1879, 182, S. 210.—Winternitz: lativ. Mitral-Insuffic. Wiener medizin. Presse, 28 März, 1880.

On reviewing the literature of the investigations which have lately en made in regard to hypertrophy of the heart, it is very evident that e attention of writers on this subject has chiefly been directed to the etiology of the disease, and that they have also limited themselves almost entirely to four leading subjects:

1. Hypertrophy caused by over-strain of the heart. 2. The connection between chlorosis and hypertrophy of the heart. 3. Hypertrophy of the heart during pregnancy. 4. The relation of hypertrophied heart to disease of the kidneys.

1. Among those who have turned their attention to "strain of the heart," the opinion appears to prevail that such a condition can exist as a cause for hypertrophy, without a mechanical hindrance being found in the circulation.

Münzinger has had unwonted opportunities for studying this question, as he has found that heart disease is very common among the peasants in the country around Tübingen, where the occupation of lifting heavy weights and climbing mountains was found combined in the vineyards where these peasants were employed. Fifty autopsies made on these people showed that, together with the changes in the heart, pulmonary emphysema and pleuritic adhesions were present in unusual amount. *Münzinger* attributes this result to the fact that the men and women, from their youth up, are obliged to carry heavy burdens on their backs, and, while doing so, were insufficiently fed. He concludes, after considering the work done by the heart and lungs of one climbing with a load on his back, that the heart and lungs are affected simultaneously, neither the emphysema, for instance, nor the heart-hypertrophy being the primary lesion, but both developing together. The symptoms do not entirely correspond with those of ordinary valvular disease. Relative insufficience predominates and weakness of the heart's action is constant, several strong contractions being followed by a preponderating number of weaker ones (delirium cordis of the French). Murmurs are not constant, and the fatal result comes from increasing debility, which is always accompanied by symptoms of pulmonary congestion.

In connection with these investigations of *Münzinger*, Prof. *Jürgensen* remarks that he never saw such affections in the lowlands.

2. *Lewinski*, after making elaborate investigations in regard to the circulatory disturbances which arise in chlorosis, precedes his exhaustive article on this subject by reporting the following case:

A boy, thirteen years of age and previously healthy, had acute articular rheumatism, which was followed by dyspnœa, palpitation, and œdema of the feet; the heart showed undoubted signs of enlargement, and there was a mitral systolic murmur and weak mitral diastolic sound, with a greatly accentuated diastolic sound over the pulmonary artery. The œdema increased rapidly, digitalis gave but little relief, and the patient died. On post-mortem examination, the left ventricle was found much hypertrophied, the right ventricle less so; the endocardium was normal, as were the bicuspid valves; the mitral orifice, however, was much distended; the aorta was unusually narrow, thin-walled, and very elastic, and the vascular branches from the descending aorta were given off irregularly; the heart-muscle was not found to be fatty, either on macroscopic or microscopic examination. No other pathological conditions having been found, the case was considered to be one of chlorosis followed by hypertrophy of the heart with relative insufficiency of the mitral valve.

He next states that *Virchow* and *Immerman* hold that, in these cases, more blood being forced into the circulation than can be accommodated by the narrowed aorta, the blood tension is increased, and the heart must hypertrophy; the later investigations on dogs, however, of *Worm-Müller* and others have shown that the amount of blood in the circulation can be considerably increased without increasing the blood tension; in chlorosis, also, the second sound of the heart is not increased, which would occur if the blood tension was increased. Then, also, if the aorta is very elastic, as is the case in chlorosis, we cannot judge of its size during life by what we find at post-mortem examination, and, as it is found that the blood has more difficulty in passing through an inflexible tube than it does where the tube is elastic, we can infer that, although the lumen of the aorta may be decreased in these cases of chlorosis, yet its elasticity being increased, and by this means the flow of blood being favored, the resulting heart-hypertrophy does not occur from increased blood tension.

Lewinski then expresses by a formula the results of his experiments on this subject: Let K represent the force of the heart and P the blood-tension; let s represent the height of a cylinder whose transverse diameter is equal to that of the mouth of the aorta and whose contents equal the amount of blood poured out by the ventricle during systole. K, however, is made up of two factors: m, which represents the whole amount of the heart-muscle, and e, which represents the individual energy of each muscular fibre. A normal circulation can then be represented by $K = P s$, or, $me = P s$; a disturbed circulation takes place when $me < Ps$. This change of equilibrium can occur in various ways; thus m may become less from chronic myocarditis, and e may become weaker from chlorosis. Chlorotic patients then may, from the abnormal condition of their circulatory apparatus, have a disease of the heart develop by which the disproportion between K and Ps may arise. Thus in aortic insufficiency, P alone is lessened, but s is so much increased that $P s$ becomes greater than K.

It is known that lessening of the blood tension in the aorta is dangerous for the economy at large, and that nature, when this lessening takes place, endeavors to obviate the danger in various ways. It is not very clear, however, why the blood pressure lessens until an equilibrium is established where the heart, that is K, is weakened, but it is known that the ability for work in the various organs of the body is dependent on the time in which a given quantity of blood can pass through the capillaries, and that the quantity is great or small, according as the function of the organ is intact or not. The celerity of the capillary circulation, other things being equal, becomes less as the aortic tension becomes less, and to avoid this lessening of celerity, various efforts at compensation are made, as increased frequency of the heart's contraction, and, most important of all, hypertrophy of the heart. Now, when $me < Ps$, P lessens, and following this, we have decreased celerity in the capillary circulation and increased functional activity of the heart and other organs; then, if e decreases still more, P again lessens, and we have what is called *a disturbance of compensation*. When, however, the status of the economy is such that it is capable of building up new tissue formations, the muscular tissue of the heart may be increased until an equilibrium is established. Chlorotic patients have this power of tissue formation in an eminent degree, and *Virchow* has shown that chlorotic individuals find that they have increased growths in various parts of their economy and in them a compensatory hypertrophy is set up to equalize the circulatory disturbances. Whether this equilibrium is ever complete from mere increase of muscular tissue is, however, questionable, for the new tissue works under the same disadvantage as the old, namely, lessened energy, or e, and hence, in spite of the hypertrophy, compensation at times does not occur. It therefore depends upon how much e is lessened, or, in other words, to what degree the decrease of the hæmoglobin in the blood is carried.

Lewinski next speaks of that class of cases where hypertrophy of the heart is found post mortem, and yet no signs of this condition are apparent during life. *Virchow* states that this often happens in chlorosis, the reason being that the usual clinical symptoms of hypertrophy are absent; these symptoms, according to *Traube*, are: 1. Abnormal resistance in the radial arteries, their walls remaining normal. 2. Accentuated aortic second sound. 3. Abnormal resistance of the heart's apex. In regard to the first symptom, increased radial tension, it often is absent where there is mitral insufficiency and an hypertrophied left ventricle, for the aortic tension is, in these cases, quite frequently normal; as to the second and third symptoms, accentuated second sound and increased force of apex beat, the left ventricle must have its strength or K increased in order to bring about these results, and, without this increased K, we cannot diagnosticate an hypertrophied left ventricle; now, in chlorosis (referring again to the formula $K = me$), the m is indeed increased, but the e is decreased, and the heart may thus be hypertrophied and yet not be more powerful, its hypertrophy apparently arising, not for the purpose of accomplishing an unusual amount of work, but only to obviate the circulatory disturbance caused by lessened energy or e of its own muscular fibres, so that the force K of the left ventricle is, at most, only normal, and hence the absence of the usual signs of hypertrophy.

Oppolzer, *Kürschner*, and others deny that relative insufficience is possible, from an anatomical stand-point, for they say that the mitral valve is large enough to close after any amount of stretching of the ostium. This, indeed, would be true if the valves, when closed, assumed a horizontal position, which is claimed by some authors, and is in accordance with *Kürschner's* manipulations, but, on referring to the latest physiological investigations on this subject, we find that this horizontal position does not occur so long as the muscles of the heart are capable of contracting. There must be a special cause for the widening of the ostium in these chlorotic cases, for, in the greatly dilated left ventricles which are found in aortic insufficiency, we often do not find a relative insufficiency of the mitral valve; there may be, perhaps, a little widening of the orifice, but nothing which in any way compares with the greatly distended mitral orifices which are found in these cases of chlorosis, where, on the other hand, the left ventricle is not nearly so much enlarged as in the cases of aortic insufficiency. This special cause *Lewinski* believes to lie in a disturbance of the papillary muscles, and he arrives at this conclusion after making a number of careful experiments on dogs.

The force of the papillary muscle equals its length, which can be represented by L, plus the energy, represented by e, of the individual fibres. Now, in the formula $K = m e$, when e is lessened, m can be increased, but this compensatory action cannot take place in the formula for the papillary muscles, for their length cannot be increased, and when e is lessened, L remains constant, and thus the force is weakened. The ventricle, therefore, does not necessarily have to be dilated for insufficience to occur, dilatation taking place when K is lessened, but insufficiency when the papillary force is decreased.

Lewinski concludes, therefore, that, through lessening of the hæmoglobin in chlorosis, the energy, e, is decreased, and the lessened force of the heart, K, is compensated for by hypertrophy or increase of m; the force of the papillary muscles, however, is lessened or lost, and from this follows widening of the ostium and relative insufficiency.

3. The literature of the supposed existence of a physiological hypertrophy of the heart during pregnancy has been a peculiar one: at a time when this occurrence was so thoroughly accepted in France that criticism was not considered necessary, it was on the contrary so little believed in, in Germany, that a direct confutation, with the exception of *Gerhardt's*, did not appear until *Löhlein* wrote on the subject in 1877, and after an examination of nine cases, found such a great variety of individual differences, that although the average of his results was rather against than in favor of a physiological hypertrophy, yet he concluded that it must still remain an open question until more extended inquiries regarding the subject were made by careful observations in the autopsy room. On reviewing the investigations of *Larcher*, *Ducret* and *Blot*, he found that the results of these authors were defective, because they had not eliminated the cases where other diseases, such as that of the kidney, existed, and he states that a heart-lesion may be masked before and then develop during pregnancy, and that during the latter months of pregnancy it is difficult to map out the boundaries of the heart. *Marty*, on the other hand, holds that a number of hindrances to the flow of blood exist during pregnancy, and considers as especially important the circumstance that the blood flows from small vessels into large canals in the uterus and that this fact, in connection with the increased extent of the vascular territory, the serpentine course of the arteries, the pressure of the uterus on the vessels, and the great increase of the portion of the vascular system lying beneath the heart, tends to render the circulation slower and to influence prejudicially the function and nutrition of the heart. *Marty* thinks that the impediment to the blood current is so

great from these causes that the heart, although it is aided by the respiration and by the contractions of the uterus, would not be able to impel the blood by its own normal strength, and in reply to *Fritsch's* statement that no hypertrophy but only an accommodation of the heart exists, he observes that accommodation is possible where a single increase in the amount of blood occurs and when this increase is of short duration; but where it is continued for months, a change in the heart must take place and in this case a simple passive increase in size is no more to be thought of. *Marty* concludes that the enlargement of the heart causes relative insufficience of the valves, thus explaining the murmurs heard during pregnancy, which as a rule disappear after delivery, the heart recovering its normal size. *Virchow* explains the occurrence of endocarditis in pregnant women by the great liability for taking cold which exists during pregnancy, and *Volkmann* and *Lah*, criticising *Marty*, show by their experiments that the work of the heart is not increased, but actually lessened by the addition to the extent of the vascular territory; but this point is not yet settled, as *Heinrich Fritsch*, on reviewing these experiments, arrived at an opposite result. *Gerhardt*, *Schroeder*, and others consider that the increased cardiac dulness, when it is found in pregnant women, is caused by a displacement of the diaphragm and lungs, and not by hypertrophy of the heart, while *Ménière* claims to have shown by numerous post-mortem examinations during the latter months of pregnancy that the walls of the left ventricle are increased from a quarter to a third in thickness. *Cohnstein*, after carefully reviewing all the results obtained by writers on this subject, arrives at certain conclusions in regard to physiological hypertrophy which are rendered especially valuable from the large number of post-mortem examinations which he has made on subjects of this class, and from the careful and methodical manner in which he has not only measured and weighed the hearts, but taken into account also the size, weight, and age of the individual, and any other possible cause which might have produced the hypertrophy in the cases where it was found. He says that pregnancy especially disposes to anæmia and chlorosis, and that the latter is admitted to be a frequent cause of enlargement of the heart. Even where the chlorosis merely consists in a change in the blood, an increase in the size of the heart has been noticed, and *Vogel* and *Wunderlich* mention enlargement of the heart following long-continued chlorosis, while *Stark* records three cases of chlorosis with enlargement of the heart, especially the left ventricle, where the enlargement passed away as the chlorosis disappeared. Now a change in the blood of pregnancy occurs, which is similar to the change which takes place in chlorosis, and thus the enlargement of the heart may arise. *Virchow* explains the valvular disease which is at times found in these cases by an especial predisposition from disturbance of nutrition. Finally *Cohnstein* states that many pregnant women, who are credited with being relatively healthy, from the fact that an hydræmic condition of the blood is accepted as a normal attribute of pregnancy, have an enlargement of the heart occur which is merely the result of a chlorosis which has developed and manifested itself during their pregnancy. These heart-changes usually appear during the second half of pregnancy. All the authorities on this subject agree that pregnancy is a dangerous complication of an existing heart lesion, by the disturbance of the equilibrium which has been established by compensation, and *Porak*, after tabulating his extensive investigations on this point, shows that of the various valvular lesions that of the mitral valve is the most dangerous, and that insufficiency of the mitral valve is attended with graver consequences than stenosis.

4. The relation between hypertrophied heart and renal disease has long been a disputed question, many theories having been bronght forward by different writers from time to time, and none of them proving to be entirely satisfactory. The earliest investigations of special importance were made by *Bright*, who supposed that the altered blood produced by disease of the kidney either directly irritated the heart or affected to such a degree the circulation in the smaller vessels that a greater activity of the heart was necessary to drive the blood through them. *Johnson* and *Traube* attributed the heart hypertrophy to mechanical hindrance in the kidney itself producing increased arterial pressure; *Traube* believing this hindrance to be due to the destruction of a large number of the renal capillaries, while *Johnson* at first supposed it to arise from a continued contraction of the lumen of the renal arterioles from hypertrophy of their muscular coat, and later imagined a state of tonic spasm of all the systemic arterioles.

The next explanation brought forward was that of *Gull* and *Sutton*, who believed that the disease was primarily a degeneration of the vascular system at large, the affection of the kidney and of the heart being a secondary manifestation, and they also stated that what *Johnson* supposed to be an hypertrophy was in reality a peculiar hyaline fibroid substance deposited in the muscular coat of the arterioles, and they therefore designated the disease arterio-capillary fibrosis.

After a minute examination of a large number of his own cases and also those of other writers on this subject, *Ewald* concludes that the kidney is the original cause of the disease, and that the vascular changes when they occur are secondary, and he supposes that the primary disease of the kidney produces a change in the blood by which the whole systemic capillary tension is increased, thus causing an increased arterial tension which is not compensated by increased activity of the vasomotors, but by hypertrophy of the heart, and that still later an hypertrophy of the muscular coats of the vessels takes place, the whole process being mechanical and not dependent on a central nervous lesion. *Senator* speaks of the obscurity in which the whole question is enveloped, arising from the fact that writers on this subject have included in their statistics all the different diseases of the kidney as a cause of heart hypertrophy, while in reality it only occurs in some, not all. He states that disease of the kidney can lead to inflammation of the pericardium, endocardium, lungs, and pleura just as valvular disease may cause disease of the kidney, and that this sequence, first observed by *Traube*, undoubtedly occurs; he then speaks of *Bäumler's* observation on the importance of the elasticity of the lung as an aid to the heart's action, especially of the right ventricle, and says that disturbance of the retractile force of the lungs must increase the work of the heart and thus produce hypertrophy perhaps of both ventricles in cases where no valvular disease exists.

Senator next makes a careful analysis of a large number of cases of parenchymatous and interstitial nephritis and arrives at the following results: "In chronic parenchymatous nephritis" "and in acute diffuse inflammation of the kidneys," "both at the beginning and at the height of the disease, the urine and its urea are lessened in amount and the blood is overloaded with urea, a condition which only occurs in the later stages of chronic interstitial nephritis. By comparing the pure forms of chronic parenchymatous and chronic interstitial nephritis, we see how greatly the influence of altered blood is overestimated and misunderstood by those who uphold this condition as a cause of hypertrophy, for in the interstitial form, where a retention of the urinary constituents does not take place, the heart hypertrophy is very commonly found, and unless some especial cause for enlargement of the cavity exists, we usually find the hypertrophy to be of the concentric variety, while in the parenchymatous form, where there is such a retention, heart hypertrophy occurs much less frequently and is so rare that many dispute its existence, and when it does occur, decided widening of the cavity or the eccentric form of hypertrophy is also found."

"If, from all the cases of chronic parenchymatous nephritis that have been reported, we eliminate all those cases where valvular disease, disease of the aorta, pericardium, lung, pleura, etc., might have caused the hypertrophy of the heart, we find that we have left very few authentic cases where this disease is found in combination with hypertrophied heart, so that we can infer that it is an accident when the two diseases happen to occur together, just as at times hypertrophy of the heart is found in connection with various other diseases of the kidney. In these few cases, above spoken of, the hypertrophy was found to be eccentric, and the disease of the kidney preceded that of the heart; and, as no other causes of hindrance to the blood current were found, there only remained, as a cause, increased aortic tension. Now, as this did not arise from circulatory hindrance in the kidney, or from increase in the amount of the blood, and as the blood was found to be overloaded with urea, we can infer that it was the urea which, by its long continuance in the blood, caused the dilatation and hypertrophy of the left ventricle, although this is not a necessary result, as the urea may be eliminated to a greater or less degree in other ways than by the kidney." *Senator* then says, in regard to the interstitial form of nephritis, that it is probably caused by the thickening of the arterial walls spoken of by authors, making more work for the heart, but that it is not proved that the primary cause lies in the kidney, and that only two causes can be thought of to explain the concentric hypertrophy: (1.) The increase of muscle outstrips the dilatation, and even where dilatation may have been present it disappears at death, being masked by the contraction of the thick muscle, this phenomenon having been noticed by *Bamberger* in cases of aortic stenosis. But in these cases the septum is usually bentover towards the right ventricle, thus giving more room in the left ventricle; and if dilatation existed, there must have been increased tension. (2.) Simple hypertrophy of the heart cannot be caused by hindrance to its being emptied, but by increase of its function and nutrition. A good example of this is the bladder, which dilates and then hypertrophies, where there is stoppage of the flow of urine, while it simply hypertrophies where there is irritation from various causes. In the case of the heart, the incentive or irritation is to be sought in the quality of the blood, which also leads to kidney cirrhosis, since the acceptance of a clearly nervous cause, as in *Basedow's* disease, is not yet proved. Finally, the disease of the kidney cannot be the cause of the increased blood tension and of the changes in the other organs, but it is only a result or partial representation of this increased pressure. *Buhl* states that the kidney and the heart are affected at the same time, and that the heart is hypertrophied early in the disease, the process of hypertrophy being, first lessened resistance of the diseased muscle to the blood-pressure, resulting in dilatation, and then, when the inflammatory process has ended, hypertrophy occurs from over-nutrition and from the increased compensatory activity of the over-distended ventricle.

In certain cases, also, the remains of inflammatory lesions of the heart lead him to believe that these lesions, from their character, must have occurred before the disease of the kidney began, and he therefore concludes that a cause as yet unknown produces a parenchymatous myocarditis and a nephritis, the progress of the disease being represented in the kidney by contraction, and in the heart by hypertrophy.

Finally, *Grawitz* and *Israel*, after reviewing the whole subject, and referring to the deeply rooted influence which *Traube's* theory of increased blood tension seems to have had with most writers, submit to a careful analysis the results of their experiments on dogs, where they were able to produce both the parenchymatous and interstitial forms of nephritis, and then to follow back each case of resulting heart hypertrophy to its cause, measuring accurately also the blood-pressure and blood-velocity under the different conditions. They speak of the great importance of recognizing the difference between eccentric and concentric hypertrophy, for they found no cases of eccentric hypertrophy or simple dilatation, excepting where, in the clinical course of the disease, an insufficient or disturbed compensation could be assumed, the animals dying from hydrothorax, ascites, etc. At times a parenchymatous myocarditis was found. In order that the compensation should be sufficient, the grade of hyperplasia of the heart must equal the defect in the kidney which remained intact, and in order that hypertrophy should arise, it was found that not only was it indispensable that the function of the kidney should be defective, but also that there should be integrity of the whole economy, and of the heart-muscle itself. The measurements showed that neither a high grade of kidney contraction nor chronic parenchymatous nephritis, nor extirpation of the kidney, even after long periods, during which heart hypertrophy had taken place, could cause increase of the arterial tension. A large amount of urea introduced into the blood did not cause increased blood tension or arterial contraction, but injections of small amounts of urea caused an increased action of the heart which, however, did not produce an increased blood tension, but an increased velocity of the blood current.

These authors then conclude that the impeded kidney secretion causes the heart hypertrophy, but that this hypertrophy does not depend on an increased arterial tension, but is caused by the small amount of urea which, being retained in the blood, excites the heart to greater activity, and thus to hypertrophy, just as it did the kidney, which in their experiments they left intact, and the function of which, as soon as it was not sufficiently compensatory, was supplemented by hypertrophy of the heart. Finally, they admit that with our present means for diagnosis, the difficulty of determining the exact time when the disease of the kidney or heart begins is so great that it is impossible to say which begins first; but they consider as proved that, under none of the conditions as yet brought forward by investigators, is the blood tension increased, and they hold that the impaired renal secretion, causing irritation and hypertrophy of the heart, with increased velocity of the circulation, produces dilatation of the vessels throughout the body.

It is thus interesting to note that the latest investigations on the subject of renal disease and heart hypertrophy concur with the suggestion made by *Bright* half a century ago, that the altered quality of the blood affords directly unwonted stimulus to the heart, causing its hypertrophy, this being the opinion held by *v. Ziemssen*, *Buhl*, *Grawitz*, and *Israel*, who concur in the belief of a disease of the kidney and heart advancing *pari passu* from impaired blood and without increase of arterial tension, also that the disease of the kidney is essentially of the interstitial form, and the hypertrophy of the heart is concentric.

IDIOPATHIC DILATATION.

BIBLIOGRAPHY.

GANGHOFNER: Die spontane Herzdilat. u. deren Folgezustand. Prager Vjhrssch., C., XXX., S. 1-54.—FOTHERGILL: The Heart and its Diseases. 1879.—BARLOW: Acute Dilatat. of Heart. Med. Times and Gaz., April 17th, 1880.

Some interesting cases have been lately reported of idiopathic dilatation of the heart, and *Fothergill* states that dilatation of the muscular tissue with enlargement of the cardiac cavities often follows conditions of passing debility and may, under such circumstances, be recovered from completely. In women, especially in those who are corpulent, the heart at times becomes weak and dilated at the menopause; no doubt the general condition may often be dependent on the feeble heart, but the reverse is also true. The following cases came under *Fothergill's* own observation.

CASE I. An elderly, hard-working woman nursed her husband for twenty days taking very little rest; at the end of this time, when seen and examined, she was found in an exhausted condition, with swollen legs, and all the physical signs of dilatation of the heart, and a rapid, feeble, irregular pulse. Under treatment all these symptoms passed away, and for the last ten years she has been a healthy woman, working as a field hand.

CASE II. A young man, eighteen years old, engaged as a field hand; the work was too hard for him, and on examination he was found to have the symptoms of a dilated heart and a weak irregular pulse. After four months' treatment with iron and digitalis all the symptoms disappeared, and he returned to work as a first-class farm servant. This happened ten years ago, and he now never feels any weakness of the heart.

FUNCTIONAL DISEASE.

BIBLIOGRAPHY.

FLINT: Functional Disorder of Heart characterized by Infrequency of Pulse. The American Practitioner, Jan., 1876. — FOTHERGILL: Neurosal and Reflex Affection of Heart. Med. Times and Gaz., Dec., 1878.—KELSEY: Functional Heart troubles. Hosp. Gaz., New York, V., VI., 1879, p. 196.

A variety of functional disease of the heart characterized by slowness of the pulse has been noticed by Professor *Austin Flint*, who concludes from his observation of six cases that it is usually associated with morbid cerebral disturbance. Of these cases there were four males and two females. The pulse varied from sixteen to twenty-six, and was intermittent in one case, irregular in one, and of normal rhythm in the others. In all the cases the cardiac disorder was recovered from, but in two the pulse remained at forty, which the author, however, is inclined to believe was not the originally normal pulse, and he thinks that it is possible to have not only a congenital slowness of the pulse, but also an acquired; the acquired, however, is of rare occurrence, although an acquired intermittency of the heart's action in middle and advanced life is not uncommon.

MYOCARDITIS.

BIBLIOGRAPHY.

RÜHLE: Zur Diagnose der Myocarditis. Deutsch. Arch. f. klin. Med., Juli 15, 1878.—ROSENBACH: Myocardit. diphtherit. Virchow, Arch., V., 70, 352.—GRISWOLD: Myocarditis, Hyper. and Dilatat. without Valvular Disease. New York Med. Jour., Feb., 1880, p. 184.

Rühle has made a careful study of the diagnosis of myocarditis, and after eliminating all those cases which occurred in connection with other diseases of the heart or pericardium, and only accepting those cases where the diseased heart-muscle itself was sufficient to explain the symptoms during life, makes an analysis of twenty-five cases observed by him, and from his results concurs in the opinion of *Köster* that these cases occur much oftener than is usually supposed, and that the most common form is the chronic diffuse myocarditis, which is frequently not diagnosticated during life, and is often overlooked at the post mortem. The favorite seat of the disease is the left ventricle, and at times it is only the outer surface of the muscular wall which is affected.

The general picture of myocarditis is that presented by uncompensated valvular lesions, passive hyperæmia, œdema, hemorrhage from various organs, great dyspnœa, a feeling of anguish on moving, and palpitation on going up-stairs, which in myocarditis occurs very early, perhaps in two or three weeks from the beginning of the disease.

The result of the muscular lesion of the left ventricle is impaired power of the heart for work, lowered aortic tension, and overfilling of the venous system. On palpation, the heart-beat is found to be of unequal force, sometimes an evident apex beat is observed, then scarcely any, then a soft flutter or a pause, and again a beat stronger or weaker than that felt at first, and thus it continues in a perfectly anomalous manner. The impulse of the apex beat can be felt as far as or even beyond the mammary line. In the neighboring cardiac area, especially in the sternal region, the heart-beat is not felt, and in the later stages of the affection it disappears entirely.

Percussion gives a constant increase of volume, as shown by the relative dulness, but this is seldom of high grade and extends to the left rather than to the right, the absolute dulness often differing very little from the normal, and at times being less than normal.

Auscultation, as a rule, gives clear valvular tones, the first sound perhaps being rather indistinct, and the second not very strong, especially in the aortic region. At times a transient systolic murmur is heard at the apex, and this was especially the case where there was a high grade of disease of the papillary muscles of the left ventricle. The force and sequence of the heart-tones are irregular, sometimes the first sound is loud, again dull, and then again a variety of tones follow quickly one upon the other and then a pause will occur, the sounds varying from quick to slow and from strong to weak. The pulse is soft, easily compressed, irregular, and unequal; the irregularity is at times so great that the number of beats in a minute cannot be stated with any certainty, and with this irregularity there is a constant inequality, the want of rhythm being even greater than that of the heart-beat.

Rühle then compares the pulse curve with that which occurs in other diseases, as pericarditis, and says that these diseases may for a time show this irregularity of pulse and heart-beat, but that this symptom soon passes away, and that where it occurs in pericarditis it is probably caused by an accompanying myocarditis.

It is at times very difficult and perhaps impossible to determine whether a chronic diffuse myocarditis is complicated or not by a valvular lesion, as sometimes a systolic murmur is heard, which does not necessarily imply a defect of the valve. Insufficiency of the bicuspid valves may be eliminated, if sternal dulness and increased pulmonary second sound are not found, and if the heart-sounds can also be heard in close proximity to the murmur.

The prognosis is bad and the most frequent cause is muscular rheumatism.

Finally, the most essential attribute in the diagnosis of chronic diffuse myocarditis is that the pulse is without any rule or regularity, and the chief symptom is the combination of irregularity and inequality, in the force of the heart's contractions and in the pulse-beat.

INFLAMMATION OF THE PERICARDIUM.

BIBLIOGRAPHY.

Weiss: Ueber die Verwachsung des Herzens mit dem Herzbeutel. Oest. med. Jahrb. No. 1, 1876.—Roberts: Paracentesis Pericard. New York Med. Jour., Dec., 1876, and Jan. 20th, 1877, and a Monograph on Paracentesis of the Pericardium, 1880.—Elliot: Case of Paracentesis of the Pericardium; Recovery. Lancet, I., 2, Jan., 1876.—Hunt: Paracentesis of Pericardium. Lancet, I., 10, March, 1877.—Hindenlang: Ein Fall von Paracentesis Pericardii. Deutsches Arch. für klin. Med., XXIV., S. 452.—Tuczek: Zur Lehre von den Pericardialverwachs. Berlin. klin. Wochenschr., 1877, No. 29.—Riegel: Pericardialverwachs. Berlin. klin. Wochenschr., Nov., 1877.—Widal: Péricardite et adhérences anciennes; paracentèse du pericarde; mort subite. Union méd., No. 138, 140.—Müller: Zwei Fälle von complicirter Pericarditis. Deutsches Archiv für

klin. Med., Dec. 24th, 1877.—Hunter: A Case of Idiopathic Pericarditis. The Glasgow Med. Jour., Sept., 1878, p. 385.—Rotch: Absence of Resonance in the Fifth Right Intercostal Space Diagnostic of Pericardial Effusion. Boston Med. and Surg. Jour., 1878, Vol. XCIX., pp. 389 and 421. Tapping of Pericardium. Boston Med. and Surg. Jour., May 20th, 1880, p. 492.—Reiss: Ueber ein neues Symptom der Herzbeutelverwachs. Berlin. klin. Wochenschr., No. 50, 1878.—Müller: Drei Fälle von Pneumopericard. Deutsches Archiv für klin. Med., Bd. XXIV., Heft 2, S. 158, 1879; and a Case of Pneumopericardium Terminating in Recovery. Med. Rec., April 15th, 1880.

The occurrence of idiopathic pericarditis has been so rarely observed, and is in fact so generally denied, that the following case reported by Dr. *Hunter* should be recorded in the literature of the pericardium:

May 27th, 1878, a girl ten years of age, previously healthy and with no hereditary tendency to disease, complained of slight pericardial pain, increased somewhat on pressure; pulse 90 and regular; breathing rather hurried. No history of cold or injury. No affection of the joints, and no evidence of any other disease, local or general, the cardiac pain being her only complaint. A superficial, harsh grating was heard in the cardiac region accompanying both the systole and diastole of the heart and not affected by cessation of breathing. No endocardial murmur was heard and there was no increase of the cardiac dulness. May 29th, the area of cardiac dulness was much increased in every direction and the friction sound not so distinct. May 31st, dulness still further increased, some cough, and considerable distress and oppression in breathing; pulse 120 and regular. June 1st, physical examination gave the following results: the dulness extended from the second to the seventh left interspace and one inch beyond the left mammary line extending also to the right, a little beyond the median line of the sternum, when the patient was in the dorsal position, and about 3 cm. to the right of the sternum when the patient was lying on the right side. An undulatory wave was seen with each cardiac impulse in the interspaces between the second and third, and third and fourth ribs. The breathing was exaggerated over the right front, and at the right base behind there was enfeebled respiratory murmur and comparative dulness on percussion. Slight cough; urine scanty and not albuminous. The apex beat, so far as it could be localized, was tilted upwards and to the right. The dulness then began to decrease and the friction sound to increase and then decrease until June 17th, when they had both disappeared, and the patient was sitting up in bed feeling much better. July 25th, the patient in the mean time being up and about, the heart-sounds were found to be normal, but the pulsation was slightly irregular. The area of dulness was normal. The apex beat was in the normal position and was rather feeble. A retraction during the heart's systole was noticed in the third, fourth, and fifth interspaces, noticeably affecting the lower half of the sternum, and remaining visible during deep inspiration.

Hunter concludes that this was a case of pericarditis with effusion, resulting in adherent pericardium.

The physical signs of a pericardial effusion at times so closely resemble those of a dilated heart, that it is no wonder that some of the most expert diagnosticians have mistaken one for the other, and, as in the case of *Baizeau* and *Roger* have tapped the right ventricle expecting to enter a distended pericardium, without harm indeed to the patients. Such a mishap is greatly to be deprecated both from the fact of a fatal case already having occurred, and also from the discredit which it may throw on an operation which has already been proved to be of great value.

It should be recognized that the usual signs laid down for the detection of effusion have frequently proved insufficient, even where the edges of the lung were freely movable, no adhesions being present to interfere with the diagnosis. *Bauer's* opinion, that the triangular shape of the area of dulness depends on the shape of the pericardial sac, has not been substantiated by later observations, the theory of *Duchek* having been found to be more correct, namely that in cases uncomplicated by pleuritic adhesions it is the retraction of the edges of the lungs which determines the shape of the absolutely dull area and that therefore the so-called triangular figure can be produced by an enlarged heart as well as a pericardial effusion. In this connection, some experiments made by producing an effusion artificially on the cadaver, where the absolute dulness was carefully noted, will be of some value as a starting point from which observers can in the future establish more precise rules for the diagnosis of this disease.

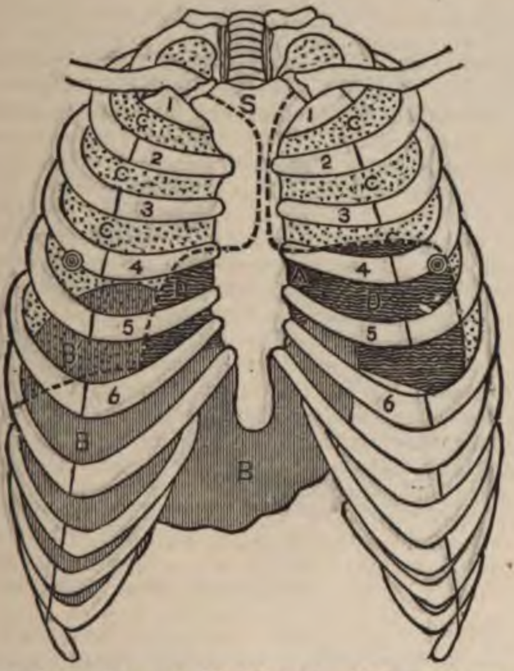


FIG. I.—Small amount of liquid introduced into sac.—ROTCH.

■ **A**—The portion of the area of absolute dulness which is still caused by the physiological dulness of the heart.

▨ **B**—Liver.

▨ $\overset{1}{B}$—That portion of the liver which is covered by the right lung.

▨ **C**—Lung.

■ **D**—Effusion.

A + D—Area of percussion dulness found when the effusion is small.

S—Sternum.

◎—Nipple.

1, 2, 3, 4, 5, 6—Ribs.

- - - Broken line—Border of lung.

The pericardial sacks of twenty subjects, sixteen infants and four adults, placed in a semi-recumbent posture and with their lungs properly inflated, were injected with melted cocoa butter, the absolute dulness marked out in ink. And subsequently, after the butter had hardened, the trachea was clamped, in order to prevent further collapse of the lung, and the sternum being carefully raised, the external inked line could by means of needles be accurately compared with the line made by the edges of the lungs with the cocoa butter cast. Different amounts of fluid were injected, and the result of the introduction of a comparatively small amount of fluid, sufficient, however, for detection by percussion, is shown in the following plate, which represents the form of absolute dulness invariably produced by the retraction of the lungs in all the cases experimented on where the sac was only slightly distended. The fluid in every case collected at the bottom of the sack and laterally on either side of the sternum, and as low as the sixth intercostal spaces; the dulness was not increased vertically, and the shape was as represented in Fig. 1.

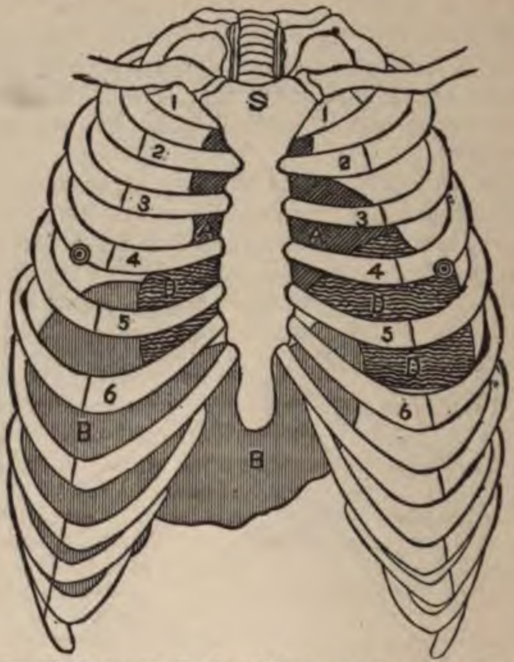


FIG. IA—Represents Fig. I. with the lungs removed.—ROTCH.

■ **A**—A portion of the normal heart enclosed in the pericardium.

▥ **B**—Liver.

▦ **D**—Effusion as it appeared in the sack, the cocoa butter being in small amount, and the lungs having been removed, after the butter had hardened.

S—Sternum.

◎—Nipple.

1, 2, 3, 4, 5, 6—Ribs.

By percussing one of the adults in the fifth right interspace, while e fluid was being introduced into the sac, absolute dulness was detected hen 70 or 80 cc. had entered. It was found impossible to make even few drops of the fluid remain at the upper part of the sac, and when e subject was inverted and the fluid allowed to harden in that position, e broadest part of the dull area was always found pointing towards the aphragm.

The only portion of the dull area, as depicted in the figure, which uld not also be caused by an enlarged heart, was that in the fifth right terspace 2 or 3 cm. from the edge of the sternum, where it would be ceedingly rare, according to authorities on this subject, to find the solute dulness of a very much enlarged heart. Hence, if the area of lness in the living subject corresponds to that found on the cadaver, solute dulness in the fifth right interspace 3 cm. from the sternal borr would be of some aid in the differential diagnosis from enlarged heart. ig. II. represents Fig. I. with the lungs removed, and it is interesting note that the upper part of the dull area is not caused by the fluid, t by the heart itself.

In connection with the results of these experiments it may be of erest to record two cases examined by several competent observers, ere the general symptoms were so clearly defined that the diagnosis s evidently pericarditis in one and endocarditis in the other, but where physical signs obtained by percussion were identical, with the extion that absolute dulness was found in the fifth right interspace in patient affected by pericarditis.

CASE 1.—PERICARDITIS.	CASE 2.—ENDOCARDITIS.
oy 6 years old.	A girl 11 years old.
ack followed acute articular rheuatism.	Attack followed acute articular rheumatism.
hopnœa, præcordial pain.	Orthopnœa, præcordial pain.
rt's impulse of about the same inensity as in case 2.	Heart's impulse of about the same intensity as in case 1.
rt's impulse felt a little to left and elow nipple.	Heart's impulse felt a little to left and below nipple.
tical absolute dulness not increased.	Vertical absolute dulness not increased.
solute dulness to left of sternum lentical with case 2.	Absolute dulness to left of sternum, identical with case 1.
solute dulness in fifth right interpace.	Absolute dulness did not extend beyond the left edge of the sternum.
doubted pericardial friction sound.	Undoubted mitral murmur.

When we consider that the friction sound is quite frequently absent ere an effusion exists, and that apparently endocardial murmurs may ur where no disease of the heart itself, but merely a pericardial effusion present, this dulness in the fifth right interspace might have proved considerable value in the differential diagnosis of the two cases; and ere the effusion is large, as in cases where paracentesis is indicated, lateral extension of the absolute dulness to the right is very evident, en the case is uncomplicated by adhesions.

In an experiment lately performed on the cadaver of an infant, the pericardium having been distended with water, two aspirator needles were introduced, one in the fourth left interspace, the other in the fifth right interspace; the fluid flowed freely from the right of the sternum, as it also did from the left, but on the left the point of the needle pricked the right ventricle as was expected from referring to Fig. II., for it is over this area and even in the fifth left interspace, unless we go well out to the left mammary line, that the layer of fluid, as shown by the cocoa-butter casts, is found to be thinnest, and at any rate is directly over the heart, while to the right of the sternum, the pericardium was found to be carried well away from the heart. This anatomical relation of the effusion and heart is substantiated clinically by *Elliot's* case, where aspiration was performed in the fifth left interspace, a little to the right of the mammary line, and, during the operation the heart was felt to strike the needle, so that it had to be directed more horizontally. An exceedingly interesting case of paracentesis pericardii is reported by *Widal*, performed by *Champenois* on a soldier, twenty-one years of age, in the hospital of *Gros-Caillou*, in 1877. The patient had been subject to attacks of rheumatism, accompanied by heart trouble for seven or eight years, and on entering the hospital for indefinite articular pains, nothing abnormal was found in the cardiac region, excepting a soft systolic souffle at the apex; some days later, however, a systolic murmur developed over the base of the heart, and on the following day, the precordial dulness was found to have extended nearly two cm. to the right of the sternum; a week later the dulness had increased still further and slight transient pericardial friction sounds were heard; four days later, examination gave the following results: the heart-beat was felt to be in its normal position; the murmur at the base of the heart had disappeared, the dulness had a transverse diameter of twenty-three cm., and extended from the heart's apex to the right axillary line, and upwards to within a finger's breadth of the lower edge of the clavicle. It did not take the form of the classical cone with its base downwards, but of an elongated ellipse, extending obliquely from left to right, and below upwards, its summit almost touching the outer third of the right clavicle. After a consultation with two other physicians, *Champenois* introduced a No. 1 *Potain* needle in the third right intercostal space, two cm. from the edge of the sternum; no fluid came out and the needle oscillated with the movements of the heart, the patient uttered a cry, made a violent effort at inspiration and died. The autopsy showed that the pericardium was adherent over nearly the whole left ventricle and at the point where the needle was introduced, the adhesions were very thick, so that the heart itself was not wounded; the pericardial covering of the right side of the heart was free from adhesions, and very much dilated, extending to the right axillary line and containing 1200 grammes of fluid; the left ventricle of the heart was hypertrophied, the right side of heart was dilated, especially the auricle, the walls of which were very thin. The heart in other respects was found to he normal. This case is instructive in reference to differential diagnosis, from the physical signs it was evident that if an effusion was present, it was complicated by adhesions, hence it was impossible to say which part of the dulness was caused by the heart itself and which by the fluid, hence it would have been safer to have tapped at a point lower down, where the heart if enlarged would not be wounded; the needle, however, was introduced, as will be seen on referring to Fig. II., just where it would be likely to penetrate the heart, and just where fluid when present is in smallest amount. One of *Hindenlang's* cases, taken from his valuable clinical review of fifty cases of paracentesis, would have been a valuable guide in the fatal case mentioned above. The patient was a waiter, twenty-one years of age, presenting symptoms of extensive pericardial effusion complicated by right-sided pleurisy. The needle was inserted in the fifth right intercostal space outside of the mammary line and twenty cubic centimetres of clear serum flowed out. When, however, the needle was pushed in deeper, a brownish-red muddy fluid escaped to the amount of 750 cc. Four days later the operation was repeated with a similar result, 550 cc. of clear pleuritic fluid being withdrawn, followed on deeper puncture by 250 cc. of red serum, which was considered to be pericardial, although it was not proved by autopsy, as the patient recovered. In opposition to the generally accepted view that paracentesis of the pericardium should only be employed where there is immediate danger to life, from the amount of the effusion or from the certainty of an unfavorable result from a purulent effusion remaining in the sac, Dr. *J. B. Roberts* has written a very careful monograph on the subject, in which, after studying the history of sixty cases with twenty-four recoveries, he states that paracentesis should be performed much oftener than has previously been done, and that we should employ it as a palliative measure just as we would tap a distended abdomen, although, perhaps, the patient might have an incurable hepatic disease. He quotes in favor of the operation *Pepper's* case, where apparent disease of the kidney was relieved by tapping an accompanying pericardial effusion; he also recommends the operation in cases of pneumo-hydro-pericarditis, and he believes that the danger from paracentesis is so slight, that more harm is liable to result from delay than from performing the operation early. The author claims that ho harm arises from repeated punctures, and his aspirating trocar with its flexible end seems to be, in connection with Potain's aspirator, the most desirable instrument for performing the operation.

In purulent effusions, *Roberts* advises washing out the cavity, and if necessary, introducing a drainage tube. The best points for tapping are thought by *Roberts* to be the fossa between the ensiform and costal cartilages on the left side or the fifth left interspace near the junction of the sixth rib with its cartilage, the latter point being preferred on account of there being less risk of puncturing the diaphragm, and he considers that *Rotch's* suggestion to tap on the right side of the sternum in the fifth interspace, four and a half to five centimetres from the edge of the sternum, must be subjected to further clinical investigation before ng accepted. In conclusion, *Roberts* states that of the above-mentioned sixty cases, thirty-six died; of these thirty-six, thirty-one had a concomitant incurable disease, and only five died from uncomplicated pericardial effusion, showing what a very favorable percentage has been obtained in this operation.

DISEASES OF THE
TERIES, VEINS AND LYMPHATICS,
INCLUDING CHYLURIA.

BY
A. T. CABOT, M.D.,
OF BOSTON,
ATTENDING SURGEON TO THE CARNEY HOSPITAL.

DISEASES OF THE ARTERIES.

MORBID AFFECTIONS OF THE EXTERNAL ARTERIAL COAT.

BIBLIOGRAPHY.

Weichselbaum: Allg. Wien. med. Zeit., 1877.—**Meyer**: Virch. Archiv, LXXIV., 1878:—**Baumgarten**: Virch. Arch., LXXVI., 1879.

Inflammation of the External Arterial Coat, Arteritis Externa, Exarteritis, Periarteritis.

Periarteritis Nodosa.

This rare disease has received much attention from pathologists since its description by *Kussmaul* and *Maier*. *Weichselbaum* and *Meyer* report cases in which most of the middle-sized and smaller arteries were covered with little nodules varying from the size of a bean to that of a grain of sand. These appeared preferably at points where small branches were given off. In *Meyer's* case, the arteries of the central nervous system were wholly free from this change, but *Weichselbaum* found the vessels of the brain similarly diseased with those of the trunk. In both cases the arteries of the heart were especially affected. On section, the nodules of noticeable size were found to contain a central cavity filled with blood-clots, and communicating with the vessel by a little opening through its wall. Besides these eccentric aneurismal sacs, both observers noticed many fusiform enlargements in the smaller vessels. After a microscopical examination of the nodules in their various stages, *Weichselbaum* came to the conclusion that the process started with a proliferation of the endothelium, and from here extended to the media and adventitia, which were both filled with round cells. The media being thus weakened, and no longer able to bear the strain of the circulation, then ruptured and so gave rise to the little aneurisms. *Meyer* found that in the smaller nodules a round-celled growth in the adventitia was the first perceptible change, and that the lumen of the vessels was slightly enlarged. The cells of the media were now indistinct and this coat became thinner, owing probably to the dilatation of the vessel. In rare instances *Meyer* found a cell proliferation also in the media. As the process advanced, the media and intima assumed a homogeneous waxy appearance, but did not give the amyloid reaction. This transparent appearance of the arterial tunics was often quite abruptly separated from the healthy parts, and occasionally affected considerable stretches of the vessel wall. The rupture of the media which gave rise to the aneurisms took place sometimes before any considerable change of the walls could be perceived, at other times thinning of the media could be made out in the neighborhood of the rent. In some of the smaller nodules, the ruptures could be seen occurring in the midst of the collections of cells above mentioned. The rents were not always complete at first, but could occasionally be seen penetrating but a part of the way through the middle coat. Sections of the aneurismal sacs showed their walls to be at times filled with round cells, and again to be simply fibrous, with but little appearance of cell proliferation. *Meyer*, noticing that the defect in the media was the one constant appearance with which the other changes were associated in varying proportions, concluded that it was the primary lesion which brought the others in its train. An examination of the muscles revealed a granular and waxy degeneration of the fibres which, though less intense than the changes observed by *Kussmaul* and *Maier*, were similar to them in character.

Baumgarten's case differed from the two just reported in the absence of the minute aneurisms, and thus agreed in this point more closely with the cases of *Kussmaul* and *Maier*. He was allowed to examine the head only. The disease was almost wholly confined to the anterior and middle meningeal arteries. He found the process to begin as a collection of small round cells in the lymph spaces between the media and adventitia. As these gathered in some quantity, the media, too, began to contain cells, and when finally the growth had attained a considerable size, the endothelium over it began to proliferate. As the growth in the outer tunics continued, the endothelial proliferation kept pace with it until the lumen of the vessel was often reduced to a little eccentric opening. Giant cells were found in all parts of these nodules, and in the larger growths fatty degeneration had commenced in the centre. The small arteries of the pia over the convexity were to a considerable extent closed by an arteritis obliterans, in which all the tunics were involved. Besides these changes in the arteries, there was a very considerable thickening of the pia mater over the posterior part of the circle of Willis, which was caused by an infiltration of round cells which showed a decided tendency to fatty degeneration, and were accompanied by giant cells. This infiltration, where it was receding, left behind it tough fibrous tissue.

Baumgarten regards this disease as a form of syphilitic arteritis agreeing in general characters with gummous inflammations in other localities. He considers that in *Meyer's* case the rupture of the media and consequent formation of aneurisms was secondary to the periarteritic inflammation, and was the effect rather than the cause of this; and he very justly points out the fact that *Meyer* did not succeed in finding a single rupture through the media of a vessel otherwise unaltered. It is also worthy of note that the aneurisms in which *Meyer* found inflammatory changes absent may well have been those in which the process was already quite old, and in which, therefore, the round cells had formed a more organized fibrous tissue.

Besides the changes in the arteries, *Weichselbaum's* patient had a general tuberculosis affecting the lungs, pleuræ, kidneys and liver, also an infarction of the spleen. One of the aneurisms upon the arteria profunda cerebri had ruptured.

In *Meyer's* case, the kidneys and liver were enlarged, and contained many of the little aneurisms which, in the kidneys, were associated with numerous small infarctions. There was also œdema of the lungs, arachnoid, and subcutaneous tissue, with a slight effusion in both pleural cavities, and in the abdomen.

SYMPTOMS.

Weichselbaum reports in his case entire loss of appetite, severe pain in the head, especially in the occipital region, tenderness over the back of the neck, vomiting and dizziness. Late in the disease hemiplegia. No albuminuria at any time. Death occurred two months after the appearance of the first symptoms.

In *Meyer's* case the duration was likewise two months. The clinical history resembled closely that given by *Kussmaul* and *Maier*. Rapidly progressive marasmus, fever, intense pain in the muscles, without, however, any paralysis, œdema of the extremities, and towards the end, albuminuria.

Baumgarten's case was only seen after the patient was moribund.

In regard to the existence of syphilis in these cases, *Weichselbaum* obtained no history of syphilis. In *Meyer's* case its existence was undoubted, and in *Baumgarten's* it was rendered extremely probable by the fact that the woman had had five miscarriages, two children who died soon after birth, and but one that survived the earliest childhood.

DISEASES OF THE MIDDLE COAT OF THE ARTERIES.

BIBLIOGRAPHY.

EWALD: Virch. Arch., LXXI., 1877.—OBERSTEINER: Oester. med. Jahrbüch., Heft II.

HYPERTROPHY OF THE MIDDLE COAT.—*Ewald* examined the smaller arteries of the pia at the base of the brain in a number of patients dying with various forms of Bright's disease. He found their walls always somewhat, often considerably thickened. This increase, he thought, depended upon a true hypertrophy of the media. He agrees with *Gala-*

bin in thinking the hypertrophy of the heart and vessels in these cases due to an increase of arterial tension caused by the resistance which the altered blood meets in the capillaries.

DISEASES OF THE INTERNAL COAT OF THE ARTERIES.

BIBLIOGRAPHY.

HEYDLOFF: Deutsch. Zeitsch. f. prak. Med., 1876.—FRIEDLÄNDER: Med. Central-Blatt, 1876.—NEELSON: Arch. d. Heilk., 1876.—SCHULZ: Ueber die Vernarb. von Arterien, etc., 1877.—BAUMGARTEN: S. g. Organis. des Thrombus, 1877.—LEGAR: Etude sur l'aortite aigüe, 1877.—MAHOMED: Lancet, II., 1877.—THOMA: Virch. Arch., LXXI., 1877.—EWALD: Virch. Arch., LXXI., 1877.—SHAKESPEARE: Toner Lect., VII., 1878.—RAAB: Virch. Arch., LXXIV., 1878.—ZAHN: Virch. Arch., LXXII., 1878.—WINIWARTER: Arch. f. klin. Chir., 1878.—PAULI: Virch. Arch., LXXVII., 1879.—SENFTLEBEN: Virch. Arch., LXXVIII., 1879.—PFITZER: Virch. Arch., LXXVII., 1879.—BAUMGARTEN: Virch. Arch., LXXVIII., 1879.

ACUTE ENDARTERITIS.

The process by which arteries are closed after ligature has been the subject of animated discussion among recent investigators. *Baumgarten*, who has published the most extensive recent work on the subject, concludes that the organized tissue closing the vessels is derived from two sources, namely: from the endothelium by proliferation, and from a growth of inflammatory (granulation) tissue, which forces its way into the vessels through ruptures at and around the point of ligature. Of these the endothelial proliferation plays the most important part. *Raab* and *Shakespeare* agree with *Baumgarten* as to the important rôle played by the endothelium in this process.

Senftleben, however, thinks that the cells noticed in the deeper layers of the intima are white blood-cells which have wandered in here, and which, undergoing further organization, supply the greater part of the tissue which finally closes the vessel. He is led to this conclusion by a long series of experiments upon animals, which it would be out of place to enumerate here. *Auerbach*, too, cannot convince himself of any active proliferation of the endothelium.

Baumgarten, in a later article, says that he has most carefully repeated the experiments of *Senftleben*, and obtained quite different results from those described; results militating in no way against his former views.

The closely allied subject of the healing of wounds in the walls of vessels has been recently investigated by *Schulz*, who thought the closure entirely effected by the organization of a fibrinous mass full of white blood-cells (white thrombus), which rapidly accumulated about the point of injury. *Pfitzer* observed the same white thrombus very soon after the injury, but found that on the third day a proliferation of the endothelium along the edges of the rent began, which finally bridged across the gap. As this bridge thickened, a similar proliferation of the adventitia took place, which, coalescing with the intima, completed the closure. The minutiæ of the changes which lead to the closure of the vessel are thus still in dispute. The truth seems to be, that the endothelium proliferates, and forms the bulk of the organized plug; also that, in some cases at least, round cells find their way in from without the vessel, to assist in the formation of the so-called organized thrombus. Whether these round cells wander in separately through intact walls, or whether they force their way in as granulations through defects in the coats of the vessel, is still an open question.

Heydloff found upon the valves and ascending portion of the aorta of a boy of eleven, who died unconscious and hemiplegic, a number of pale-red granulations, which were composed of round cells, and grew from the intima. There was also in places a round-celled infiltration of the inner coat.

Zahn gives the name endarteritis verrucosa to a condition found by him in a man of thirty-five, who died of tuberculosis. In the aorta and in the common and external iliac arteries, were a number of little wart-like growths, some of which attained the size of a pea. They consisted of collections of cells between the two layers of the intima, and showed a strong tendency to fatty degeneration.

Leger describes as acute aortitis a process coming on secondary to the chronic atheromatous change, and in which all the coats of the aorta are inflamed. The symptoms are feelings of oppression in the chest, burning behind the sternum, paroxysms of pain like angina pectoris, various murmurs in the course of the ascending aorta, with increased cardiac dulness. The disease is usually of several months' duration.

Liebermeister thinks these cases should be included under the head of atheroma, as the duration of the disease would class it with chronic rather than with acute processes.

ARTERITIS OBLITERANS.

Under this name *Friedländer* describes a process which, having its starting point in the intima, may properly be classed under the head of endarteritis.

The intima of the small and middle-sized arteries is infiltrated in places with small cells, causing a uniform or one-sided narrowing of the vessels. A similar infiltration of the adventitia coincides with the appearance of these cells in the intima, and *Friedländer* thinks that some of the latter may come from without. These cells finally organize, and form fibrous tissue. This process he noticed in induration of the lungs, and of other organs, and in the neighborhood of tumors. *Pauli* examined vessels closed by proximity to cavities in the lungs, and agreed in the main with *Friedländer's* view of the process, but found the infiltration of the media and adventitia very inconsiderable. *Baumgarten*, on the other hand, thinks that the process starts in the adventitia, and later affects the intima.

Thoma noticed a process similar to that described by *Friedländer*, in the middle-sized arteries of kidneys affected with chronic interstitial nephritis. The larger renal arteries in these cases he found often atheromatous, or even calcified. In parts also of these larger vessels, the connective tissue lying between the muscle cells of the media had proliferated (mesarteritis). In the smallest branches of these same vessels he found the intima occupied by a hyaline, slightly granular mass, lying next the media, on which it pressed, and which was somewhat atrophied; the adventitia, on the other hand, was thickened, and merged without sharp boundary line in the interstitial tissue about. This latter condition corresponds with what *Gull* and *Sutton* describe as arterio-capillary fibrosis, and these observations of *Thoma* indicate some connection between this and other endarteritic affections.

Ewald, also studying the renal vessels in interstitial nephritis, says that he finds the hyaline changes only in the smallest arteries without either adventitia or intima. In the vessels of more considerable size, he finds the principal changes in the intima, which is first infiltrated with young cells and later becomes fibrous. The media is sometimes, though not often, atrophied and the vessels are narrowed. In this connection some observations of *Neelson* are of interest. He found the capillaries, in fifty out of seventy-nine brains examined, affected by a change similar to this just described. Their walls were thickened and transparent for short stretches, particularly at points where they branched. The process started in the endothelium. The nuclei became shrunken, and then the cells were changed into a hyaline mass which bulged out into the surrounding lymph spaces. This change he found almost invariably in the brains of old persons, more rarely in the middle-aged, and almost never in children.

Winiwarter reports a case of arteritis obliterans of the tibial arteries which caused gangrene of the foot requiring amputation. The patient was a strong man of fifty-seven, whose legs were constantly wet and chilled and who had for years suffered from pains in his feet. An obliterating endophlebitis had also closed the veins, and the nerves of the leg were sclerosed.

Mahomed thinks a fibrous thickening of the smaller vessels very common in persons of advanced age, when many tissues (cartilage, arachnoid, etc.) take on a fibrous character. In such cases he believes it often to be independent of any affection of the kidneys. The only symptom by which the disease makes itself known is a hard, resistant pulse, with increased arterial tension shown by the sphygmograph. This is, of course, useless for diagnosis unless all other causes for such increased tension can be eliminated.

SYPHILITIC DISEASES OF THE ARTERIES.

BIBLIOGRAPHY.

Baumgarten: Arch. d. Heilk., 1875.—**Friedländer**: Med. Central-Blatt, 1876.—**Köster**: Berl. klin. Wochenschr., 1876.—**Baumgarten**: Virch. Arch., LXXIII., 1878.—**Huber**: Virch. Arch., LXXIX., 1880.

Friedländer points out the fact that the endarteritis described by *Heubner* as characteristic of syphilis is exactly similar to the arteritis obliterans which has been shown by *Cornil* and *Ranvier* and by himself to occur in the course of many diseases not syphilitic. He adds, however, that this process has hitherto been found primarily only in cases of syphilis.

Köster asserts that the nodules upon the intima always correspond with inflammatory collections of cells in the other coats which coincide in position with the points where the nutritive vessels break up into capillary tufts, and further, that this form of disease only occurs in vessels supplied with vasa vasorum.

Baumgarten agrees with *Friedländer* in thinking the process described by *Heubner* identical with arteritis obliterans. He thinks that the proliferation of cells in syphilitic arteritis commences in the adventitia and only affects the intima later in the process. *Baumgarten* cannot, however, agree with those who think that there is but one disease affecting the vessels of the brain, "arterio-sclerosis," under which a syphilitic form is to be ranked. He believes that syphilis of the arteries is a distinct affection, and advances the rule that in arterio-sclerosis you have an endarteritis which tends to degenerative changes, and is accompanied by a proliferating peri- and mesarteritis, while in syphilis of the arteries a peri- and mesarteritis tending to degeneration is the primary change and is accompanied by a proliferating endarteritis. (See further under periarteritis nodosa.)

Huber reports the case of a girl of twenty-two in which the history of syphilis was unquestionable and which offered the following appearances post-mortem. Besides many inflammatory conditions in various parts of the mucous surface, there was extensive amyloid degeneration of the abdominal organs and an affection of the vascular system described as follows: The arteries of the extremities and abdominal cavity showed the disease in its fullest development; the arteries of the lungs were less affected, and those of the heart and brain not at all. In the veins were similar changes, though less in degree. The deeper layers of the intima were first filled with round and star-shaped cells, later the media contained round cells, and still later the adventitia. The endothelium was unchanged over the top of the growth. The cells in these growths soon became fattily degenerated and finally led to calcification. The early stages of this process were found in the larger arteries, as little gray or yellow elevations grouped often around the point where a branch was given off; as one advanced towards the smaller branches the process increased in extent, involving the whole calibre of the artery, which was, in many places, transformed into a resistant calcified tube.

Huber also mentions a case in which a similar calcification of the arteries in the neighborhood of several gummata was observed, the vessels throughout the rest of the body being unchanged.

ANEURISM.

BIBLIOGRAPHY.

Maunoury.-Jean: Bull. de la Soc. Anat. de Paris. 1875.—**Schrötter**: Allg. Wein. med. Zeit., 1876.—**Jawerthal**: Riv. clin. di Bologna, Dec., 1876.—**Ponfick**: Virch. Arch., LXVII., 1876.—**Colles**: Dublin Journ. Med. Sc., LXIV., 1877.—**Weir**: Am. Journ. Med. Sc., LXXVII., 1879.—**Barwell**: Med. Chir. Trans., LXII., 1879.—**Weir**: Arch. of Medicine, N. Y., III., 1880.

PATHOLOGY.

Multiple Aneurisms.—*Ponfick* reports a case of multiple aneuisms of the gastro-epiploic and gastric arteries. They were mostly small, but one reached the size of a pigeon's egg. Death resulted from the rupture of the largest of these upon the left gastro-epiploic artery. They were shown to be of embolic origin, the emboli coming from an endocarditis of the mitral valve. *Jean* saw a case in which the ascending portion and arch of the aorta were dilated into a fusiform enlargement, on the walls of which were three little aneurismal sacs.

Maunoury saw many little eccentric sacs along the thoracic aorta. The wall of the vessel was covered with atheromatous patches. (See also under periarteritis nodosa.)

SYMPTOMS.

Mahomed gives as the sphygmographic signs of aortic aneurism: (1) Diminished elevation of pulse wave. (2) Less tension. (3) Diminution or abolition of dicrotism. (4) In some cases a number of waves following the primary elevation. (5) Postponement of pulse on one side as compared with the other.

Schrötter and *Jawerthal* call attention to the fact that in aortic aneurism a considerable pulsation at the lower part of the trachea may be observed with the laryngoscope.

Buchwald reports the physical signs in a case of aneurism of the trunk of the pulmonary artery. They were: a wavy pulsation in the second intercostal space to the left of the sternum, and a systolic murmur extending into diastole and best heard over the pulmonary valves.

TREATMENT.

Barwell has collected the cases of ligature of the right subclavian and carotid for aneurism of the innominate. They are eleven in all, and three of them resulted successfully. He also reports one case in which he tied these same vessels for aneurism of the arch of the aorta, the patient left the hospital improved. He selected the right carotid and subclavian, because the circulation in them is less intense than in the vessels on the left, consequently their closure would throw less strain back upon the aneurism.

Weir tabulates twenty-two cases in which the rubber bandage was used in the treatment of aneurisms of the extremities. A cure was obtained in twelve cases. In nine cases the attempt was unsuccessful and one case died. He concludes that the bandage is a very good addition to the means which we now possess of arresting the circulation in the aneurism, and lays stress upon the importance of allowing the sac to be full, and uncovered by the bandage, which envelops the limb above and below.

It is best used in alternation with the tourniquet, each being left on two hours at a time till a cessation of pulsation is brought about. The one fatal case occurred in a man with fibroid phthisis and fatty heart.

Schwalbe considers that the advantage derived from injections of ergot about an aneurism is due to the irritant action of the ingredients in the preparation used, which strengthens the sac by promoting induration about it.

OBLITERATION OF THE ARTERIES.

BIBLIOGRAPHY.

PREISENDORFER: Obliteration of the Innominate Artery almost complete, obliteration of the left carotid and subclavian with aneurism of the arch of the aorta and carcinoma of the œsophagus. Virch. Arch., LXXIII., 1878.—STIFEL: Am. Jour. Med. Sc., LXXIX., 1880.

Stifel tabulates forty-six cases of closure of the pulmonary artery. In six cases, the trunk of the artery was a solid cord. In thirty, the orifice of the artery was closed. In ten, the exact point of closure was not mentioned. In thirty-five cases, there was an opening in the septum ventriculorum. In thirty-eight cases, the ductus arteriosus was permeable, in four it was closed. In twenty-eight cases, the foramen ovale was open, in four closed, and in fourteen not noted.

DISEASES OF THE VEINS.

ENDO-PHLEBITIS.

See case of *Winiwarter's* under Arteritis Obliterans.

BIBLIOGRAPHY.

Langhans: Virch. Arch., LXVII., 1879.—Schwalbe: Virch. Arch., LXVI., 1879.—Duret: Arch. gén. de méd., LXXX., 1879.

Dilatation of the Veins.—*Langhans* saw a boy of nineteen, who for ten years had suffered from hæmaturia, of which he finally died. The veins about the fundus of the bladder were much dilated, and several small cavernous tumors projected beneath the mucous membrane. Ruptures in several of these had caused the fatal hemorrhage.

Duret ascribes the production of hemorrhoids to three causes:—1. The physiological acts which cause an increase of tension in the portal vein, such as forcing at stool. 2. Mechanical obstruction to the flow of the blood through the portal vein, as frequently results from congestion of the internal organs, especially the liver. 3. Spasmodic contraction of the sphincter, preventing the flow of blood through the anastomosing branches, which connect the hemorrhoidal veins with the branches of the internal iliac vein outside of the rectum. When the second cause is suspected, he recommends cold douches over the hepatic and splenic regions, and claims much benefit in many cases from this method of treatment.

Schwalbe treats varices by injections of alcohol in their immediate neighborhood, and reports good results, owing, he thinks, to the increased growth of connective tissue thus induced.

DISEASES OF THE LYMPHATICS.

INFLAMMATION OF THE LYMPHATICS.

BIBLIOGRAPHY.

Schmidt: Pflüger's Archiv.—Lassar: Virch. Arch., LXIX., 1877.—Curtis: Boston M. and S. J., CII., 1880.

Schmidt finds that in diffuse suppurative inflammations the lymphatics become stiff and inelastic, and are filled by firm clots. In gangrenous inflammation the secretion of lymph is almost wholly stopped.

Lassar, experimenting upon animals, found that if a limb was acutely inflamed by the application of croton oil, the lymphatics carried from it ten to twenty times as much lymph as normally. This lymph stream was equally abundant above and below a gland, even if the gland was inflamed. The lymph, from a part thus inflamed, differed from normal lymph in being thick and sticky, and containing great quantities of indifferent cells.

Curtis describes sudden swellings of the lips, which, appearing in a night, disappear with like rapidity, rarely lasting forty-eight hours. This enlargement is generally painless, though sometimes accompanied by slight tenderness. There is some heat and occasionally redness of the part. No cause can be assigned for this affection. *Curtis* ascribes it to an acute reticular lymphangitis.

NARROWING, OCCLUSION, AND DILATATION OF LYMPHATICS.

BIBLIOGRAPHY.

WEGNER: Arch. f. klin. Chirurg., 1877.—BUSEY: Amer. J. Obstet., 1877-78.—LANGHANS: Virch. Arch., LXXV., 1879.

Wegner has examined carefully a number of cases of lymphangioma. He divides this group into three classes: simple, cavernous, and cystoid lymphangioma. The first includes those forms which are made up of a network of somewhat dilated lymph-vessels and spaces. The second class comprises those tumors consisting of many communicating cavities of small size, and containing lymph either fluid or coagulated. This lymph thrombus may undergo colloid changes, and may contain red blood-cells, pigment, granular cells, fat drops and crystals. The general structure of this form resembles that of the corpus cavernosum. In the third class the cystic dilatations may reach a considerable size, and communicate but imperfectly with each other and the lymphatic trunks. Familiar examples of lymphangioma are the congenital forms, macroglossia, macrocheilia, macromelia, and hygroma of the neck. Other favorite seats for these conditions are the forehead, shoulders, nates, extremities, axillæ and inguinal regions; they also appear occasionally in the internal organs. They usually originate in the subcutaneous fat, and in the loose connective tissue between the muscles and around the large vessels. They may present diffuse or circumscribed forms, and in the former the skin covering them often takes on the appearance familiar in elephantiasis. If they press upon blood-vessels, these may become dilated, and the pressure gradually causing atrophy of their walls, a communication may be established between the blood and lymph channels, forming a hæmato-lymphangioma.

The etiology of these tumors is still obscure. They probably depend upon closure or obstruction, and consequent dilatation of the pre-existing lymph-vessels and spaces (ectasia with hyperplasia). In one case, *Wegner* thinks he found a second method of development, as follows: The endothelium of the lymph-channels proliferated, forming a mass of cells, in which new channels and cavities were formed (homoplastic neoplasia). As a third possible method is mentioned, the formation of lymph-vessels and spaces in granulations growing from a connective tissue matrix (heteroplastic neoplasia).

Langhans injected the lymphatic trunks in a case of lymphangiectasis of the leg, and was unable to discover any closure or narrowing.

SYMPTOMS.

These tumors are usually soft, painless, and unaccompanied by general symptoms; they are, however, subject to recurring inflammatory attacks, during which they swell, become red and tender, and are accompanied by fever. They at times come to a stop in their growth, and may even recede.

By firm pressure the simple and cavernous forms may be partially emptied of their contents, and slowly resume their form upon the removal of the compression. The cystoid form cannot generally be so compressed. A rupture of one of the distended cysts may lead to a permanent fistula, through which lymph is constantly being lost.

Busey calls attention to the increase in growth of such tumors as affect the female genitals during the menstrual periods.

Lebert speaks of the anæmia which comes on in course of a lymphorrhagia. He calls it a spoliative anæmia, thinking that the elements of the blood are destroyed more rapidly than they are produced.

CHYLURIA.

BIBLIOGRAPHY.

MANSON: Jour. Lond. Linn. Zool. Soc., XIV., 1878.—MANSON: Med. T. and Gaz., I., 1878.—RONCIERE: Arch. de méd. navale, 1878.—COBBOLD: Lancet, II., —COBBOLD: Parasites of Man and Animals, 1879.—FAYRER: Lancet, I., 1879. Y. Med. Jour., XXXI., 1880.

urine is white like milk, or may have a pink tinge from It usually coagulates in about half an hour, but, his coagulum again liquefies and a cream rises to the surface. Prof. *E. S. Wood* has allowed me the use of three unpublished ases in which he made careful quantitative analyses of the urine, one of which I reproduce here as a sample.

Total urine for twenty-four hours,	1750 cc.
Specific gravity,	1021
Total amount of urea,	44.625 gms.
Total amount of chlorine,	8.273 gms.
Total amount of phosphoric acid,	3.570 gms.
Total amount of albumen,	2.489 gms.
Total amount of fat,	5.229 gms.

In this case the difference between the urine passed before and after food was well shown.

Before Breakfast.

Amount passed,	135 cc.
Specific gravity,	1024
Amount of fat,	0.324 gm.

After Dinner.

Amount passed,	138 cc.
Specific gravity,	1030
Amount of fat,	1.501 gm.

In most cases the quantity of urine is increased. In Dr. *Wood's* cases the daily amount varied from 1660 cc. to 2180 cc. Sometimes there is marked suppression. *Abbe* reports such a case in which a girl of 18 passed but 96 cc. of urine daily, with a specific gravity of 1026.

ETIOLOGY.

This disease has been shown to depend, in the majority of cases, pon the presence in the blood of the minute parasites discovered by *Wucherer* and *Lewis* and named Filaria sanguinis hominis.

The fact that chyluria occurs sometimes in patients who have never isited the tropics where these parasites abound, and that none can be iscovered in their urine or blood, renders it probable that in rare cases he disease may depend on some still undiscovered condition unconected with the entozoa. Besides chyluria, many other diseases indigeous to the tropics are traced to this same source. Of these I would ame elephantiasis, lymph-scrotum, craw-craw, some cases of leprosy, æmaturia, phlebitis, furuncles, hydrocele with fibrinous or chylous fluid.

Persons with filariæ in their blood may have several of these affections t one time, or they may follow each other at irregular intervals. The endency to a recurrence of these diseases persists for long periods of ime, and after years of good health the parasites have been found in the lood of persons once affected.

Manson asserts that one-eighth of the population of Amoy have filariæ in their blood. *Hall* and *Patterson* found them in one-twelfth of the people examined in India.

Opinions differ concerning the natural history of these parasites. In 1878, *Manson* in Amoy found that, when a mosquito drew blood from a filarious patient, it sucked up a great number of filariæ, out of proportion to the amount of blood drawn. Following the history of the parasite in the stomach of the mosquito, he found that about one in ten survived and increased in size; that soon an alimentary canal and other markings appeared, and that finally this more matured parasite was thrown out into the water when the mosquito discharged its ova.

Manson considered this period of growth in the mosquito a sort of chrysalis stage, and believed that the animal then, entering the human system either through the skin or the alimentary canal, underwent its final development and fecundation within the body. *Bancroft*, in 1877, first discovered the adult parasite. It was a thin, hair-like worm, three or four inches in length. One specimen he obtained from a "lymphatic abcess" of the arm, four others from hydroceles in chyluric patients.

Other observers reject *Manson's* system of development on the ground that they find the filariæ to be digested in the mosquito's stomach. *Manson* replies that nine-tenths are so digested, but that the other one-tenth survive, and further adds that these observers have undoubtedly mistaken a filaria affecting dogs for the filaria sanguinis hominis, and says that this canine parasite is indeed digested in the mosquito's stomach.

PATHOLOGY.

The observations upon the changes in the organs in chyluria are very scanty, as the disease does not tend to a fatal termination. Dr. *Lewis* reports an autopsy in which filariæ were found throughout the capillaries of the kidneys. The lymph channels lying beside the urinary tubules were also found dilated.

SYMPTOMS.

The genetic connection of phlebitis, elephantiasis, etc., with chyluria, is of importance in considering the symptomatology of this disease. *Abbe* saw one chyluric patient suffering from furuncles and phlebitis, and mentions another who, within the space of a few years, had chyluria, several attacks of erysipelas, craw-craw, lymph-scrotum, and scrotal elephantiasis.

In two of *Abbe's* cases, extreme prostration existed, disappearing simultaneously with the return of the urine to a normal condition. One of *Wood's* cases suffered from pain in the bladder, rectum, and perineum, especially after rowing.

TREATMENT.

Prophylaxis.—Late investigations point to the water used for bathing or drinking as the probable vehicle by which the parasite obtains an entry into the system. All water used in regions where the disease abounds should be boiled and filtered before use. In regulating the diet for chyluric patients, it is worth remembering that a tolerably exclusive use of vegetable food diminishes the amount of chyle passed.

As this is probably due to the diminution of pressure in the lymphatic vessels, it may be a directly curative measure by allowing defects in the walls of these lymphatics time to close.

In the way of medication, tonics are sometimes of use, as also astringents, especially gallic acid. In several cases turpentine has hastened the cure.

DISEASES OF THE STOMACH.—DISEASES OF THE INTESTINES.—DISEASES OF THE SPLEEN.

BY

ELBRIDGE G. CUTLER, M.D.,

OF BOSTON,

VISITING PHYSICIAN TO CARNEY HOSPITAL; PHYSICIAN TO OUT-PATIENTS MASS. GEN. HOSPITAL.

STOMACH.

PRELIMINARY PHYSIOLOGICAL REMARKS.

BIBLIOGRAPHY.

GRÜTZNER: Neue Untersuchungen über die Bildung und Ausscheidung des Pepsins. Breslau, 1875, S. 82.—**KRETSCHY**: Deutsch. Arch. f. klin. Med., XVIII., p. 527.—**EDINGER, L.**: Das Verhalten der freien Salzsäure des Magensaftes in zwei Fällen von amyloider Degeneration der Magenschleimhaut. Berlin. klin. Wochenschr., No. 9, 1880.—**VELDEN, R. VON DEN**: Ueber die Wirksamkeit des Mundspeichels im Magen. Deutsch. Archiv f. klinische Medicin. December, 1879, p. 105.—**HEIDENHAIN, R.**: Ueber die Absonderung der Fundusdrüsen des Magens. Arch. f. d. ges. Physiol. Bonn, 1879, XIX., 148–166.—**EWALD, C. A.**: Weitere Beiträge zur Lehre von der Verdauung. Zeitschrift für klin. Medicin, Bd, I., pp. 614-630.

Ebstein and *Grützner* have recently found that more pepsin may be extracted from a pylorus by glycerin, if the pylorus has previously been properly treated with common salt, for the latter frees the pepsin from its combination with the albuminates (so-called pepsinogenous substance). This fact, and the circumstance that the gastric juice contains a quantity of chlorides, led *Grützner* to the investigation "whether it was the chlorides in the body which separated the pepsin from the albumen-holding ('principal') cells, and appeared with it on the free surface of the stomach." The experiments, which were performed on the dog and the pig, showed that *the quantity of chlorides in the mucous membrane both of the pylorus and fundus was subject to certain slight variations, which were dependent on the abundance of pepsin, so that large secreting* ("principal") *cells contain more chlorides than small shrivelled ones.* For example, the mucous membrane, dried and rich in pepsin, from the fundus of a dog fasting forty-four hours contained one and four-hundredths per cent of NaCl, that of a dog killed six hours after feeding on sponge, the "principal" cells being very small and poor in pepsin, was only sixty-two hundredths per cent. Of especial interest was the result of an experiment in which 250 cubic centimetres of a four-per-cent solution of common salt were injected into the jugular of a dog that had fasted forty-two hours. In an hour the dog was killed, and at the autopsy the "principal" cells did not appear great and clear as might have been expected in the fasting condition of the animal, but small and shrivelled, and corresponding to it the quantity of pepsin of the stomach was diminished; that is, the NaCl on the mucous membrane of the stomach freed the pepsin from the "principal” cells, and thereby brought about on the mucous membrane of the fasting animal an appearance corresponding to a condition of the stomach which has normally secreted for several hours.

Brücke's assumption, that the *cause of the expulsion of the chyme* is to be found in the irritation of the walls of the stomach by the increasing acidity which occurs towards the end of digestion, has received confirmation through the recent very interesting observations of *Kretschy.* His experiments, which were carried out on a person affected with a gastric fistula, showed that acid gastric juice flowed out of the stomach fistula for six and a half hours after an abundant meal; *further that in the sixth hour the maximum acidity was gradually reached, in the seventh hour a rapid fall to a neutral reaction occurred,* and with it complete emptying of the stomach took place.

As a further very noteworthy result of the investigations mentioned, we may briefly say: during the *menstruation* of the patient, *digestion was extremely protracted,* so that with three meals within twenty-four hours, only in the twenty-third and twenty-fourth hours (in the morning between five and seven o'clock) did the fluid from the gastric fistula have a neutral reaction. The exhibition of alcohol and of coffee exercised a retarding influence on the course of digestion; 0.6 to 0.8 gms. of pepsin given a quarter of an hour before dinner did *not* hasten the digestion of the food taken.

Free hydrochloric acid has hitherto been found constantly absent from the gastric juice only in one disease, dilatation of the stomach dependent on stenosis from carcinoma of the pylorus. *Edinger* has seen it temporarily absent during the existence of a gastric catarrh, accompanied by a great secretion of mucus, and in the dyspepsia occurring in the course of febrile diseases (especially typhoid fever). He found it to be always absent on frequent examination in two cases where the arteries of the mucous membrane of the stomach had undergone amyloid degeneration.

GASTRITIS.

BIBLIOGRAPHY.

GRIESINGER: Klin. u. anat. Beobachtungen, etc. Archiv für physiol. Heilk., Bd. XIII., S. 528, 1854.—TROUSSEAU: Vertigo dyspeptica. Gaz. des Hôpit., 1862; Schmidt's Jahrb., 117, 34.—GALLICIER: Du vertige stomacale. Thèse, Paris, 1866; Virchow-Hirsch's Jahresber., II., 129.—STEWART: Gastritis phlegmonosa. Edinb. Med. Journ., February; Canstatt's Jahresber., II., S. 125, 1868.—LOOMIS: On Suppurative Cellular Inflammation of the Stomach. N. Y. Med. Record, 1870; Canstatt's Jahresber., II., 156.—BELFRAGE and HEDENIUS: Upsala lökaref. förhdl., VII. Ref. Schmidt's Jahrb., 1872, S. 298.—VON BASCH: Ueber den Magenschwindel u. verwandte Zustände. Wien. med. Presse, 1873, Nr. 15 ff.—SUSUM SATO: Ueber Durchfall bei Kindern. Diss. inaug., Berlin, 1874.—RAKO-

WAK: Ein Fall von Gastritis submucosa. Wiener med. Presse, Nr. 25, 1874.—LÖWENSTEIN: Ueber Gastritis phlegmonosa. Diss. inaug., Kiel, 1874.—FAGGE, HILTON: A Case of Diffused Suppurative Inflammation of the Stomach. Transac. of the Pathol. Soc., XXVI.; Virchow-Hirsch's Jahresber., 1875, II., S. 225.—GRÜTZNER, P.: Neue Unters. über die Bildung und Ausscheidung des Pepsins. Breslau, 1875.—MOSLER: Weitere Erfahrungen über den Nutzen der Einführung grösserer Flüssigkeitsmengen in den Darm. Deutsch. Arch. für klin. Med., XV., S. 233, 1875.—GLAX, J.: Ueber Gastritis phlegmonosa. Berlin. klin. Wochenschrift, 1879, XVI., 565–568.—DEININGER: Zwei Fälle von idiopathischer Gastritis phlegmonosa. Deutsch. Arch. f. klin. Med., Band XXIII., 624–632.—LEWANDOWSKY, H.: Zur Casuistic der idiopath. Gastritis phlegmon. Berliner klin. Wochenschr., No. 38, 1879, p. 568.

Leube speaks of the astonishing result of the use of calomel in cholera infantum, and says he commences treatment by the use of this drug without exception. The first day it is given in cathartic doses, and at the same time the only food allowed is a little meat-broth. The next day he commences the diet mentioned in Vol. VII., p. 152, and follows out other treatment directed to the inflammation of the intestinal tract and increased peristalsis.

Ipecacuanha has been frequently recommended, and is to be given either in powder, gms. 0.025 to 0.1 at a dose or in the form of clyster (infusion ipecac., gms. 0.5 to 5.0 in 100 gms. of menstruum). It is possible that the ipecacuanhic acid, which has an analogous action to tannic acid, may be chiefly efficacious here. When the diarrhœa persists a long time, astringents may be added to the hydrochloric acid already recommended, Vol. VII., p. 151, with a view to stopping it.

℞ Extr. gentianæ,	
Acidi hydrochlorici (dil.),	
Acidi tannici	āā 0.5
Aquæ destillatæ	40.0
Syrupi althææ	20.0

M. A teaspoonful every two hours till the diarrhœa is stopped (for a child six months old altogether three to five teaspoonfuls).

Recently *Mosler* has obtained good results in enteritis by *irrigation of the intestines* with a five-per-cent solution of permanganate of potassium repeated frequently during the day.

GASTRITIS PHLEGMONOSA.—Of the four cases reported by *Glax* and *Lewandowsky*, there were autopsies in but two, so that the accuracy of the diagnosis must remain doubtful in the other two, especially as one of them recovered. All the patients were males, aged seventeen, forty-six, fifty, and fifty-two years, respectively. The chief symptoms in each case were sudden, severe vomiting, and pain in the abdomen, with great prostration. Only in the case which recovered was pus vomited; in the others bile-stained fluid was brought up. One case began with a severe rigor, and the temperature rose, during the seven days the patient lived, to 40.4° Cent. (104.7° Fahr.), with a pulse of 136, and respirations 36. In the case that recovered, the temperature for eight days ranged between 39° and 41° Cent.

(102.2° to 105.8° Fahr.), with a very rapid pulse, and the whole course of the illness, which lasted a month, much resembled typhoid fever. In one case there was delirium, in another great restlessness, with a feeling of suffocation. In one of Dr. *Glax's* patients, there was considerable enlargement of the liver and spleen, and he also had general peritonitis, with a good deal of inflammatory effusion into the abdominal cavity. The stomach in this case was the seat of diffuse purulent infiltration of its walls. In the second case that was examined post mortem, the infiltration was much less marked, and was partly purulent, partly serous. Its anterior and posterior surfaces were of a dark-violet color, and small punctate hæmorrhages were scattered here and there over the mucous membrane. In two of the four cases, the diseased condition may have been due to errors of diet (one patient had eaten large quantities of unripe grapes); in the third the patient was a hard drinker; in the fourth no assignable cause could be discovered for the fatal illness, and Dr. *Lewandowsky*, the family physician, to whom the patient had been known for years, could remember no previous attack of any kind which might have been the starting-point of the final catastrophe. In 1878, *Leube* had only been able to collect thirty-five reported cases of phlegmonous gastritis, including examples of the diffuse and the circumscribed forms. Dr. *Lewandowsky* thinks the total number of cases described up to 1879 but slightly exceeds forty. No doubt, however, it has not always been recognized. The diagnostic points which separate it from gastritis, gastro-enteritis, and circumscribed peritonitis, are, according to *Deininger* (*Deutsches Archiv für klin. Med.*, Bd. xxiii.), three in number: First, the fever and general symptoms are much more severe than in these diseases; secondly, the pain is not aggravated by the patient's movements; thirdly, the gastric region offers a feeling of very much increased resistance. The diffuse form runs a more rapidly fatal course than the circumscribed form (gastric abscess); the latter may drag on for months, and kill the patient at last by exhaustion and slow fever. The treatment is at present, we need scarcely add, utterly unsatisfactory. Dr. *Glax* agrees with *Deininger* that the early use of cold gives the most rational chance of success in this direction.

DILATATION OF THE STOMACH.

BIBLIOGRAPHY.

Korn: Ueber die Pylorusstenose. Diss. inaug., Königsberg, 1868.—**Habershon**: Guy's Hosp. Rep., XVI., p. 399. Virchow-Hirsch's Jahresber., 1871, II., 148.—**Wald**: Ueber Magenektasie. Diss., Berol., 1872.—**Jacoby**: Ein Fall von

Verschluss des Pylorus. Berliner klin. Wochenschr., 1872, Nr. 38 (bei einem Tuberculösen). — STRAUSS: Ueber Magenerweiterung. Diss., Berlin, 1873.—WINTERNITZ and BAUM: Die Magenerweiterung. Wiener med. Presse, 1873, Nr. 17.—FAGGE, HILTON: On acute Dilatation. Guy's Hosp. Rep., 18. Virchow-Hirsch's Jahresber., 1873, II., S. 155.—BIERMER: Ueber Magenerweiterung, etc., ibid., 1874, II., 248. Correspondenzbl. für Schweizer Aerzte, Nr. 2.—OSER: Die mechanische Behandlung der Magen- u. Darmkrankheiten. Wiener Klinik, 8, 1875.—GRÖTSCHEL: Ueber Magenektasie. Diss., Berol., 1875.—VON BASCH: Ueber Magenerweiterung. Wiener med. Presse, 1875, Nos. 20 and 22.—LEUBE: Zur Diagnose der Magendilatation. Deutsches Archiv für klin. Med., Bd. XV., S. 394. —EKBERG: Toa Fall af Dilatatio, etc. Upsala Läkarefören. förh., Bd. X., 414. Virchow-Hirsch's Jahresber., 1875, II., S. 227.—ROSENBACH: Zur Diagnose der Magenerweiterung. Deutsche med. Wochenschr, Nos. 20, 21, 1876.—FERBER: Ein Beitrag zur Lehre von der Magenperkussion nebst Notizen über Magenektasie. Deutsche Zeitschr. für prakt. Medicin, 1876, No. 42.—PURGESZ, Dr. S.: Zur Diagnose und Therapie der Magenerweiterung. Deutsches Arch. f. klin. Med., Bd. XXIII., p. 554–563.—ALLBUTT, Dr. C.: Use of the Stomach-pump in treating Gastric Dilatation. Brit. Med. Journ., Feb., 1880, p. 315.—SNEDDON, Dr. W.: Dilatation of Stomach successfully treated by the Syphon Stomach-tube. Brit. Med. Journ., Jan. 1880, p. 51.—ROSENBACK, OTTOMAR: Der Mechanisms und Diagnose der Mageninsufficienz. Sammlung klinischer Vorträge, Volkmann, No. 153.—EBSTEIN, W.: Incontinentia Pylori. Sammlung klin. Vorträge, No. 155.

Purgesz determined the amount of entrance of the sound into the stomach by the following method. He fastened a manometer to the outer end of the sound, and proved that a negative pressure was always to be read off, changing with the phases of respiration so long as the sound was in the thoracic portion of the œsophagus; as soon as the sound passed the hiatus œsophageus of the diaphragm, however, a change of pressure instantly appeared in the manometer, and instead of the former negative, a positive pressure was found, varying in amount with the phases of respiration. Since the moment of this change in pressure, that is the entrance of the sound into the cardia, can be readily observed, we have favorable conditions for determining the diameter of the stomach; it only is necessary to mark the place where the change in pressure has occurred, and then push the sound carefully forward till we reach an obstacle, or till the lower end of the sound can be felt through the abdominal walls. If the sound is graduated, we have only to subtract the number obtained when the sound is fully introduced from that where the change to positive pressure occurs, and the result is the vertical diameter of the stomach.

In a case under *Leube's* observation, which recently came to autopsy (carcinoma of the pylorus), the pylorus alone was dilated, so that the shape of the stomach was quadrilateral.

In explaining the occurrence of gastrektasia in drinkers, we must, in addition to the large quantity of fluid usually taken by such people, also take into account the *quality* of the drink, as the latest experiments of *Kretschy* have shown that the introduction of alcohol into the stomach has a retarding influence on digestion.

Ferber has recently proved that there appears on the posterior wall of the thorax, in the region of the posterior lower border of the lung, a tolerably great dulness, reaching variably high, within which pectoral fremitus and respiratory murmur are almost null, but which reappear again, however, with disappearance of the dulness, when the patient assumes the knee-elbow position. *Ferber* explains the appearance as follows: in the upright position of the patient, the tense abdominal muscles press the stomach upwards and backwards, so that high position of the diaphragm and retraction of the lung result.

Rosenbach has lately proposed an ingenious way of diagnosticating insufficiency of the expelling power of the stomach. He passes a sound into the stomach and ties a rubber tube ending in a balloon on the free extremity. When the balloon is squeezed, if the eye of the sound is below the level of the fluid in the stomach, one hears with the ear over the abdominal wall a "coarse bubbling, moist, often metallic râle, with a distinct splashing of fluid," while in case the eye of the sound does not dip into the fluid, either nothing is heard, or, only a hissing noise. With the aid of this experiment, and taking into account the length of the piece of sound in front of the incisor teeth, we may determine, by pouring in and pumping out, the position of the fluid in the stomach, and from its behavior in these manœuvres draw a conclusion as to the contraction and distention of the stomach. *Rosenbach*, from the results of his experiments, makes the deduction that by pouring in large quantities of fluid (500 to 1000 cc.) in healthy persons there is a rise; in sick people, in whom the elastic and muscular power of the stomach has become insufficient, under certain circumstances, a constancy, or even a sinking of the level as a result; on pumping out, on the other hand, in such patients a sinking of the level occurs, which is deeper than corresponds to the amount of fluid removed.

Recently *cold rubbing* has been advised in the treatment of dilatation of the stomach, and *Winternitz* and *Baum*, in using it in conjunction with dry diet, have obtained good results which they attributed to the procedure having a tonic influence on the relaxed muscular tissue.

NERVOUS DYSPEPSIA.

BIBLIOGRAPHY.

LEUBE, W. O.: Ueber nervöse Dyspepsie. Deutsches Archiv für klin. Medicin, Dec., 1878, p. 98.

It is a well-known fact that in many healthy people gastric and intestinal digestion have a perceptible influence on the general feelings; and though the latter, after a moderate dinner or supper, are not essentially altered, yet, after every hearty meal, in a large number of people symptoms appear which manifest themselves as a feeling of slight discomfort.

There is quite frequently the sensation of fulness in the epigastrium, congestion of the head, slight excitement, besides disinclination to mental labor, a tired feeling, especially in the legs, and sleepiness.

There can be no doubt that these phenomena are *the effect of the act of digestion on the nervous system.* How this effect is brought about it is difficult to say. An explanation may be sought in two directions, according as we regard the influence of the nervous system through gastric digestion as the result of the *chemical* changes which occur, or as the purely *mechanical* effect of the ingestion of food. With regard to the chemical explanation, we might presuppose that the resorption of the products of digestion in the stomach might exert a moderate irritation on the nervous system, and thus be the cause of the series of symptoms mentioned above. This would be analogous to the influence which the resorption of *abnormal* products of digestion may exercise on the general nervous system under certain circumstances. Since the observation of *Senator* (*Berliner klin. Wochenschrift,* 1868, No. 24, Ueber einen Fall von Hydrothionämie und Selbstinfection, etc.), where in a case of acute gastric catarrh, on the second day of the disease, eructation of sulphuretted hydrogen, dizziness, quickening of the pulse, and collapse showed themselves, and sulphuretted hydrogen appeared in the urine, it is at least not improbable that the general disturbances of the nervous system which accompany acute and chronic gastric catarrh, as headache, dizziness, palpitation, weariness of the limbs, in short, which appear as general depression of the bodily and mental functions, depend on the resorption of products of abnormal digestion. Butyric acid and similar abnormal products of digestion which are formed under the influence of gastritis might cause poisoning of the central nervous system which would find expression in these nervous appearances. Similar but weaker actions on the nervous system would be produced by the normal products of gastric digestion, as lactic acid, and thereby we obtain an explanation for the occurrence of the above-named nervous symptoms appearing in healthy people after meals, especially the general fatigue and sleepiness (*Preyer, Centralblatt für die med. Wissensch.,* 1871, p. 577).

Plausible as this reasoning may appear for the explanation of the question, yet it has great, indeed, insurmountable difficulties. First, it is a matter of daily experience that the nervous appearances following a hearty meal *do not by any means first occur in the course of digestion,* and keep pace with the collection of the digestive products; but for the most part *they occur immediately after eating,* as for example, the languor which is insurmountable in some individuals, and only disappears after the usual noonday nap. How could this *rapid* occurrence of the nervous symptoms be brought in accord with a poisoning in the above sense, without doing great violence to matters of fact ? How can it be explained that the nervous appearances are already fully developed before the chemical transposition of the ingested food *can* have occurred in any considerable quantity, and, on the other hand, just at the height of the process of digestion disappear again ? How would it be quite conceivable that by a short sleep, often lasting only a few minutes, congestion, fatigue, sensation of fulness, and so forth, in short all discomfort should disappear without leaving any traces, or that in another person a cup of tea, or coffee, etc., suffices to banish the nervous appearances for all the rest of the period of digestion? Lastly, it would be least of all strange that, in case the resorption of poisonous matters were the cause of the trouble, the quality of the food were not of great weight in determining the severity of the symptoms in question, while, as a matter of fact, the quality comes into account only incidentally, and the quantity of food ingested is that which seems to play the chief rôle.

From all this it does not seem possible to regard a chemical action of the digestive process on the nervous system as the essential thing in these disturbances; it appears more plausible rather to regard them as the result of the *direct mechanical irritation which the gastric nerves suffer at the commencement of digestion.* The most natural explanation appears to be that the entrance of the stomach on digestion, *the commencement of complete activity of the stomach, is not without powerful reaction on its nerves and secondarily on the entire nervous system.* Probably besides the sudden change of the distribution of blood, mechanical stretching and irritation of the walls of the organ play an important part, and perhaps a part of the change in the general reaction of the nervous system is to be directly attributed to the sudden changes of blood-pressure, such as *Mayer* and *Pribram* (*Sitzungsberichte der Wiener Academie,* 1872, Abth. III., Band II.) were able to produce experimentally by mechanical irritation of the wall of the stomach.

These nervous appearances accompanying digestion and still within physiological limits may obtain pathological significance *when they occur in unusual severity,* without the cause lying in the manner of the ingestion of food. They form then a very prolific source of complaints and may finally, by becoming permanent, *cause a severe chronic affection whose source is to be sought in an abnormal reaction of the gastric nerves, and secondarily of the whole nervous system in stomach digestion.* The increased excitation and irritability of the nerves of the stomach during digestion, therefore, in such cases do not form a symptom, but the true essence of the disease to the description of which we shall presently pass, and which may be designated as *difficult digestion of nervous origin,* or better still as *nervous dyspepsia.* As an independent disease this must be sharply separated from the nervous appearances which prevail in gastric catarrh, cancer of the stomach, etc., since they here represent nothing more than one of the different symptoms by which the pathologico-anatomical well characterized disease of the stomach in question is distinguished clinically.

If we go back to the cause which occasions this pathological increase of the participation of the nervous system in the digestive act it must be sought in two directions: either the character of the irritation which the nerves of the stomach meet with is an abnormal one, or the nerves of the stomach themselves are in a condition which makes them react excessively or abnormally to a normal irritation. Such an abnormal sort of irritation, coming under the first category, may be regarded as the most important cause of the nervous dyspeptic symptoms in cases of severe disease of the stomach, in gastric catarrh, ulcer of the stomach, cancer, etc., in short in all the organic diseases of the stomach. In consequence of the structural change of the wall of the stomach, its function becomes abnormal; the expulsion of food is delayed, the transposition of ingesta is not regular in that the gastric juice secreted by the diseased gastric wall, abnormally composed, yields abnormal products of digestion. The result of this is that the nerves of the stomach are influenced too long and in addition by unusual irritants during digestion, whence no doubt the feeling of mental and bodily depression, sleeplessness, dizziness, etc., in short, a part of the well known history of dyspepsia is to be traced. *Here we must consider as essential the dependence of those phenomena on the digestion rendered abnormal by the gastric disease in question;* they are nothing else than symptoms of gastric catarrh, cancer, etc., which indeed in some cases may come very prominently to the front, especially when in the course of the disease the nutrition of the patient in question suffers.

The matter is different when digestion does not last pathologically long, when apparently no abnormal chemical products are furnished by it, and, nevertheless, the nervous symptoms of difficult digestion appear, and completely master the whole clinical picture; in other words, when *these dyspeptic appearances have their basis only in the nervous system and its abnormal irritability,* while the gastric wall as well as the gastric juice is unchanged, and accordingly the digestion with regard to time and chemically runs an essentially normal course. It is conceivable that we may succeed in the future in finding anatomical changes in the nervous system also in those cases of dyspepsia coming exclusively from the nerves of the stomach, just as we must cling to the same hope in regard to neuralgias and different neuroses in spite of the usual negative appearances at autopsy. *We must only designate as "nervous dyspepsia" that type of disease in which anatomically nothing stands in the way of assuming an exclusive participation of the nervous system, especially of the nerves of the stomach, in the occurrence of the dyspepsia.*

Leube says the disease is by no means rare. He has had an opportunity of observing a whole series of cases in the last few years. Almost without exception, the disease occurred in people of the higher circles of society, and both sexes were about equally affected. The age of the patients was mostly mature youth, the first decade after puberty. In considering the cases, that which strikes the attention most is *the disproportion between the great subjective complaints of the patient on the one side, and the objective result of the* (jeweiligen) *occasional digestive act on the other.*

In two cases, sounding and washing out the stomach seven hours after a prescribed meal gave perfectly clear wash-water, with no trace of mingled food; which showed that the expulsion of the contents of the stomach was fully accomplished in the usual time, and that the digestive power of the stomach accordingly, at least as regards its duration, was normal. Notwithstanding this, there appear a whole army of complaints which stand in evident relation with the digestion—congestion and headache, fatigue and sleepiness after eating, swelling of the gastric region, sensation of fulness in the epigastrium, irregularity of appetite, annoying eructations. The latter is like the other symptoms mentioned to be regarded as a nervous appearance according to *Leube*. It always occurs when the nerves of the stomach are powerfully irritated.

DIFFERENTIAL DIAGNOSIS.

The clinical picture in nervous dyspepsia is wholly different from carcinoma ventriculi, when the latter does not run an extraordinarily latent course. The rapidly increasing cachexia, the greater age of the patient, the painful sensations in the gastric region, the vomiting which occurs with tolerable regularity in the course of the disease, the appearance of the vomited matter, and finally a tumor appreciable to the touch, give strong evidence of cancer, while only the dyspeptic appearances are common to cancer and the disease we treat of. There are cases of cancer of the stomach which show almost none of the above symptoms, where the patient feels perhaps merely languid and miserable, and only slight dyspeptic appearances indicate that a chronic disease of the stomach, is at the bottom of his troubles. In such rare cases the diagnosis of carcinoma is impossible and the differential diagnosis lies between cancer, nervous dyspepsia, and chronic gastric catarrh.

The essential difference between these two diseases of the stomach, in my opinion, is that in chronic gastric catarrh loss of appetite is a prominent symptom, abnormal decompositions of the digestive products occur in the stomach, the ingesta stay longer in the stomach than normal and are usually vomited, ordinarily with large quantities of mucus; while on the contrary, in nervous dyspepsia, the period of digestion runs its course in the normal limits, the food is completely expelled from the stomach, and mucus is wanting in the vomited matter. We may also be permitted to make a probable diagnosis of the existence of a nervous dyspepsia, when that which had been hitherto regarded as chronic gastritis remains unaffected by carefully conducted dietetic treatment. This probable diagnosis is established when a test washing-out of the stomach proves that there is normal digestion and a treatment directed less to the digestion than to the general nervous condition is followed by decided improvement.

In many cases the differential diagnosis between nervous dyspepsia and ulcus ventriculi is more difficult. In the latter, it is known that the digestion, as such, may occur in the normal limits of time without disturbance of the appetite or vomiting, and the pain may be very slight, or even entirely wanting. If such patients complain of pressure in the epigastrium, eructation, nausea, headache, and sleepiness, the diagnosis becomes more difficult as examination with the sound in such cases of ulcus ventriculi may show normal digestion of the stomach. In such a position which speaks with far greater probability for the existence of a nervous dyspepsia than for an ulcer, we resort to therapeutics. We assume, in the first place, that those slight nervous dyspeptic symptoms are not caused by an idiopathic perverse reaction of the nerves of the stomach, but by a more or less latent ulcer of the stomach. We therefore place the patient on a rigid diet, let him eat only meat solution and a little milk for ten days or two weeks, keep him in bed and put warm compresses over the body, and in short maintain the regimen which has proved so efficacious with us for about six years in every case of gastric ulcer. If the trouble does not decidedly improve under this treatment—as it usually does—we exclude ulcer, and treat the disease as nervous dyspepsia, according to the principles to be given below. Just as the failure of a rigorous dietetic treatment indicates nervous dyspepsia in doubtful cases, so does conversely the favorable action of an application of electricity to the gastric region *during* digestion. If the fulness and pressure in the epigastrium disappear during this application, when this happens in full digestion, it is highly improbable that a gastric ulcer is the cause of those nervous dyspeptic symptoms. In the majority of cases of ulcer of the stomach, the clinical history, however, is cleared up by one or another symptom coming out prominently, whether it be a severe pain dependent more or less on eating, and on the position of the patient, or pain in the dorsal region, or bloody vomiting, or a consecutive gastrectasia, which makes the diagnosis undoubted.

What was said above as characteristic of chronic gastric catarrh in contradistinction to nervous dyspepsia applies also in the main to *dilatation of the stomach*, though this happily, at least in pronounced degrees, cannot well escape physical examination, and therefore presents less difficulties for a differential diagnosis. Just as little also does *gastralgia* have to be considered in practice, for the neuralgic paroxysmal character of the affection is sufficient for the diagnosis. This is absent in nervous dyspepsia; the immoderate irritability of the nerves of the stomach is here rather a chronic condition which shows itself in every irritation affecting the nerves as a sign of dyspepsia. In the pure forms of gastralgia, on the other hand, the latter fail; it represents rather a neuralgia in the narrower sense, in which periods of severe pain alternate with periods of freedom from pain, in which the patient feels perfectly well.

With regard to the *prognosis*, we must say that, in our experience, it is by no means very favorable. Cure is certainly not impossible, yet, in most of the cases, we have been forced to be contented with partial success. With regard to complete recovery, nervous dyspepsia is exposed to the same great variations as the other nervous diseases of a general nature, and we might remark again that just this disease in question has proved a very stubborn evil.

Corresponding to the nature of the disease, the *treatment* must be directed first towards strengthening the entire nervous system. Apart from influencing the will of the patient by moral dietetics, a cold-water cure is always chiefly indicated. For drugs we have used quinine and iron. Besides the daily application of douches and packs, we have regularly given electricity, partly in the form of very powerful induced currents, partly as the constant current. The latter is used sometimes externally and sometimes internally. In the external application the anode is placed on the epigastrium, the kathode in the region of the vertebræ, and ten to fifty elements employed, according to the sensibility of the patient. The internal application was performed according to the method given by *Beard* and *Rockwell*. In general, the latter method has not been strikingly more efficacious than the external application of electricity, so that, for the past year, we have not used the internal galvanization of the stomach.

In regard to the *diet*, in general we do not allow food either which is difficult of digestion or hearty meals, in order to avoid immoderate action of the nerves of the stomach and by the extreme quiet given by a careful regimen get back more and more to a normal reaction. It is not necessary for us to give a fixed diet-list here. Ergotin exhibited in one of our cases proved of very great service.

In the subsequent treatment, sea baths, and, in delicate constitutions, residence among the mountains is indicated.

DISEASES OF THE INTESTINES.

PHYSIOLOGICAL CONSIDERATIONS.

By the precipitation of pepsin in the duodenum, a further important effect is produced, to which *Kühne* has recently called attention. Since trypsin (the albumen-digesting enzyme [ferment] of the pancreatic juice, *Kühne*, 1. Sitzungsber. des naturhistor. med. Heidelberger Vereins, N. S., I., 3, 1876) is destroyed by pepsin, its action would be impeded and pancreatic digestion disturbed, unless the pepsin were precipitated by the bile. By this is explained the favoring influence of bile on the pancreatic digestion, as well as the increased necessity for food in animals with biliary fistula.

ENTERITIS. INTESTINAL CATARRH.

BIBLIOGRAPHY.

CHOUPPE, H.: Note sur l'emploi de l'ipecacuanha, etc. Virchow-Hirsch's Jahresber., 1874, II., 251.—PUYGAUTHIER: Sur l'emploi de l'oxyde de zinc dans le traitement de la diarrhée. Thèse, 1874, ibid., 1875, II., 227.—MOSLER: Weitere Erfahrungen über den Nutzen der Einführung grösserer Mengen von Flüssigkeit in den Darmkanal, etc. Deutsches Archiv für klin. Med., XV., S. 223, 1875.

The most important therapeutic measure in intestinal catarrh is washing out at stated intervals. As in the treatment of catarrh of the stomach, frequent washing out of the diseased organ, at least in the beginning of the treatment, is the best means of removing the products of decomposition which keep up the inflammation; so in inflammation of the intestine, a washing out of that is demanded, since here more than in the stomach is opportunity presented for fermentation and decomposition. There is also less danger of removing nutritious matter than in gastric catarrh. For regularly washing out the intestine *Leube* uses, after *Hegar's* recommendation, a simple funnel with rubber tubing and a nozzle; the fluid injected is usually a weak carbolic acid solution (one part alcohol, one part carbolic acid, five hundred parts water). As a rule, it is a matter of indifference whether the injection is made with the patient on the side or on the back; but in cases where it is desirable to send the fluid as high up as possible, it is well to place the patient on the side with the buttocks raised, so that the intra-abdominal pressure may be diminished, though for this purpose the knee-elbow position, troublesome to many patients, must be acknowledged to be the surest.

Ipecacuanha in dose of 0.12 grammes, or in clyster 20 to 500, evaporated to 250, is frequently used.

ENTERALGIA.

BIBLIOGRAPHY.

CASTAN: Relation d'une épidémie de colique sèche. Montpellier médical, Mars, p. 189; Virchow-Hirsch's Jahresber., 1873, I., 248.—FRANK: Ueber die Pulsveränderungen bei Bleikolik. Berliner klinische Wochenschrift, No. 9, S. 118, 1875.—REISSLAND: Ueber Bleikolik. Berliner klin. Wochenschrift, S. 284, 1875.

In *epidemic* colic, the cause of the epidemic is naturally to be found in the action of injurious substances on a large circle of individuals. These injurious matters, in the large proportion of cases of colic epidemics, are poisonous substances, especially lead preparations, which are conveyed to a great number of people at the same time, in tobacco, flour, water, and so forth. Yet certain epidemics seem to have arisen without the action of those well-kown toxic substances.

Thus *Castan* reports an epidemic which occurred in Montpellier in the late summer and autmn of 1872, raging only in the southern part of the city and its vicinity, which was exposed to marshy influences, the course of the disease resembling lead colic. All investigations as to the source of any possible lead poisoning were in vain, so that in this case nothing remained but to refer the cause to cold and malaria.

Recently *Frank*, from observations on eighty-five patients with lead colic, *has determined a sphygmographic curve peculiar to the pulse in this disease.* The chief characteristics of it are: great retardation in the fall of the descending-line, especially of the terminal portion of it, strongly marked elasticity, relatively small dicrotic rise, approach of the secondary ascent on the top of the curve, and, in pronounced cases, a peculiar top-curve, consisting of two notches.

In the *treatment* of enteralgia, continued irrigations may be of great service, as in the case of *Riessland* (l. c., p. 286) where the most severe colicky pains completely disappeared on the repeated injection of several litres of water, opium having failed.

DISEASES OF THE SPLEEN.

ANATOMICAL AND PHYSIOLOGICAL REMARKS.

The view of *Bichat* and *Meckel* that the size of the spleen increases during digestion has been confirmed by *Giesker* (Anatomisch-physiologische Untersuchungen über die Milz des Menschen. Zürich, 1835, p. 41) and *Gray* (On the Structure and Use of the Spleen. London, 1854) experimentally.

The weight of the spleen, according to *C. Krause*, amounts on the average to 225 grammes (between 210 and 315). The volume is determined by *Krause* at 221.5 cubic centimetres. The relation of the weight of the spleen to the weight of the whole body remains stationary from birth to middle life (1 to 320 or 400).

The envelope of the spleen is formed by the blending of the peritoneal covering with a fibrous layer of its own. From the hilus the capsule of the spleen, for a short distance, can be dissected into a fibrous and a serous layer. Its thickness amounts to 0.08 or 0.2 mm. It is formed of interlacing bundles of connective tissue, which gradually become thinner from without inwards. In the deeper layers, besides elastic and connective tissue fibres, there occur scanty bundles of smooth, muscular fibres (*Henle*). These latter have also been found by *Meissner*, *W. Müller*, *Schwarz*, *Kyber*, and *Waldeyer*. *E. Klein* has recently confirmed their occurrence (Observations on the Structure of the Spleen. *Quart. Journ. Microsc. Sc.*, p. 363, October. Virch. *Jahresb.*, 1876, Bd. I., S. 54) in small bundles, both in the capsule and in the trabeculæ of the spleen in dogs, apes, and human beings. In the human subject, the bundles in the capsules are less numerous, they are chiefly present only where the trabeculæ branch off; the latter, both great and small, always contain numerous bundles of smooth, muscular fibres (*Klein*).

Next to the capsule, the substance of the spleen, the parenchyma, is of special interest to the physician. By washing out or macerating it can be reduced to two structures—a denser framework, which resists maceration, and a soft pulp-like mass, which is contained in the meshes of the above. The framework is partly formed of branching blood-vessels, and partly of fibrous cords which run inwards from the capsule, connecting frequently with each other and with the walls of the vessels, and consisting of the same elements as the capsule. The soft mass, the pulp of the spleen, in the broader sense, shows, in the fresh or hardened state, likewise two constituents, which are distinguished from each other by their color, and somewhat also by their consistency. In a uniform red groundwork, *the pulp in the narrower sense or red pulp*, are imbedded gelatinous white bodies, mostly of globular shape, the splenic follicles or corpuscula Malpighii. On cross section the latter appear as circular figures of 0.03 to 0.5 mm. diameter, more rarely as short streaks, in whose centre or axis the section of a blood-vessel appears.

The primitive branches into which the artery and vein of the spleen divide at the hilus, six to twelve in number, do not anastomose with each other, and in the interior of the spleen keep separate. The organ is thus divided by its blood-vessels into tolerably regular portions, and the pathological anatomist gets a distinct idea of them through certain peculiarly defined affections. From the isolation of the different vascular districts, one may convince one's self by injection that from an arterial branch always only a certain portion of the spleen can be filled. The injection returns by the corresponding venous trunk, and does not spread into the adjoining vascular territory (*Giesker*, *Gray*). The last sort of expanding of the splenic arteries is the so-called penicilli. Every penicillus, with its corresponding vein, forms a closed vascular system. The spleen is consequently divided into a number of single parts, or, if one may say so, lobes and lobules. In splenic hypertrophy the lobar form often appears. The splenic follicles stand in a close relation to the finest arterial branches. Each arterial and venous trunk enters the spleen in a common sheath, which is the direct continuance of the capsule, and they divide at first in common. When the sheath and the vessels, by repeated division, have become very small, the course of the vessels separates, and at the same time the sheath of the artery suffers a modification which gives rise to the formation of the splenic follicle. We may designate this modification, according to *Henle*, as a transformation of the loose connective tissue into conglobate tissue, a diminution and fibrillation of the bundles to a delicate reticulum, and filling of the interspaces of the reticulum with cells resembling lymph-corpuscles, which are held together by a more or less tenacious connective substance. The follicles are globular, here and there rather elongated tumefactions of this sheath. If the tumefaction is one-sided, the follicle hangs rather laterally on the artery, and sometimes is even pedunculated.

The capillaries of the conglobate substance are few. In the follicles they come from small arterial branches, which enter from without or from the traversing artery. Having emerged from the conglobate sheaths, the small arteries break up quickly into diverging branches, which, when they are drawn out of the soft substance, become tufted or brush-shaped (penicilli arteriarum). These branches consist of the true capillary tube and an adventitia.

If the veins are followed from the spot where the arteries have left the common sheath, one sees that they branch arborescently; the larger branches run straight, or are slightly curved; the finer ones in irregular and angular curves. The mutual relation of the last arterial and venous twigs calls to mind the relation of the hepatic and portal branches to each other, in that the venous branches on section form starlike figures with abundant branches running out from them, in the circumference of which figures are the arterial branches (*Billroth*).

The veins issuing from the sheath have, besides their endothelium, at first a slight layer of connective-tissue, which is distinguished by an abundant layer of cells. On the finer and finest branches of the capillary veins of *Billroth*, the endothelium, as well as the connective tissue layer, suffers a peculiar transformation. Instead of the rhombic plates of the first, there appear relatively small spindle-shaped cells, pointed at both ends, with a nucleus placed about in the middle of the length of the cell, and bulging out from the same. The cells project into the interior of the vessel, as is seen on cross section.

The most difficult and disputed point in the anatomy of the spleen is the transition of the blood from the capillary arteries into the capillary veins. Instead of the venous sinuses described by *Billroth*, *W. Müller* supposes an *intermediate blood-channel* of the pulp, a system interposed between the capillary vessels and the commencement of the veins, in which the blood moves in channels without walls. From the intermediate channels the blood is again collected in the broken commencements of the venous system, since the walls of the venous radicles proceed from the pulp-tissue, and thus the blood comes into close relation with the elements of the pulp. The doctrine of blood-channels without walls in the parenchyma of the spleen was first expressed on the ground of injection preparations of *von Stieda*, then in *W. Müller's* monograph it was carried through the spleen in all classes of vertebrates. Since then, authorities of great weight have added their opinion in confirmation—*M. Schultze, Henle, Waldeyer, Frey*, and others.

The conception of the splenic pulp is, therefore, dependent on the ideas concerning the blood-channels of the spleen. *W. Müller* reckons with the splenic pulp, besides the elements peculiar to the spleen, the blood-corpuscles sometimes lying outside the closed vessels, the number of which, together with the amount of blood plasma found in the passage between the arteries and veins, is changeable, and which, through its inconstancy, is the chief cause of the variations in the size of the spleen.

The tissue elements peculiar to the spleen are cells which are not essentially different from those of the conglobate substance described above, and a part of which are always increasing (*W. Müller*); and a delicate reticular intercellular substance. The splenic framework is regarded as a breeding-ground for numerous leucocytes and giant-cells (*Klein*). Besides the regular elements of the spleen, the parenchyma contains, in varying quantity, as accidental constituents, the different shades of pigment, from yellow to dark-brown, partly in single molecules, partly in clumps, numerous transition forms between colorless and red blood-corpuscles, and the cells containing blood-corpuscles.

With regard to the lymphatics of the human spleen, an injection of them has not yet been perfectly successful. Their relation, therefore, has been judged chiefly by analogy from the results obtained in animals, mostly in the horse (*Tomsa*), concerning which we must refer to special works.

The nerves consist, in the great majority of cases, of organic fibres. At first they lie in the same sheath with the arteries and veins, and accompany the arteries in very fine branches. *Ocken* (Inaugural dissertation. Greifswald, 1875), in his dissertation, prepared under the direction of Dr. *A. Budge*, proved that the axis cylinders of the non-medullary nerve-fibres of the spleen may be split up into fibrillæ by reagents.

Concerning the functions of the spleen, opinions are still more divided than concerning its anatomical arrangement. It is admitted on all sides that it has an influence on the formation of blood. On the ground of his anatomical investigations, *W. Müller* has attributed to it the function of continual new formation of colorless blood-corpuscles, and their constant addition to the blood current. He compares the structure of the spleen to that of a lymph gland, in which the lymph stream is replaced by the blood current. Physiological experiments, as well as pathological facts, make it probable, moreover, that the spleen forms not only a breeding-ground for colorless blood-corpuscles, but that the transition from white to red blood-corpuscles (which former are known to be the source of origin of the red blood-corpuscles in extra-uterine life) takes place in the spleen just as in the medulla of bones. Also the belief has been entertained that the spleen must be regarded as a place where red blood-corpuscles disappeared, upon which disappearance depend the pigment and the iron found in this organ.

Diligent observers have been at work, especially of late, to obtain more accurate results concerning the function of the spleen. Different methods of investigation have been used, and many difficulties have been met with, which may perhaps serve to explain the contradictory results. One of the first and most important methods of investigation was the comparison of the blood of the splenic artery and that of the vein; also, later, of the splenic lymph with that of other parts of the body. In this respect we have already positive results: for the blood, the relative predominance of the colorless corpuscles in the venous branches; for the lymph of the spleen, the admixture of colored blood-corpuscles, peculiar to it alone, which, as *Tomsa* has shown, ceases with death, and is also probably dependent on the pressure under which the parenchyma of the living spleen stands. Opposing observations also exist in regard to the character of the venous blood of the spleen (*Torchanoff* et *Swaen*, *Archiv de physiol. norm. et path.*, 1875, p. 324). According to *Robin* (Diction. Encyclop. des Scienc. Médic.), the spleen exercises an influence only on the chemical composition of the blood and lymph plasma, but not on the relation of the morphological elements in the blood. Concerning the chemical relations, we possess important information which deserves mention here: the pulp of the spleen has an acid reaction; it contains, besides all the constituents of the blood, manifold products of oxidation, hypoxanthin, xanthin, uric acid, leucin, tyrosin, inosit, volatile fat acids (formic, acetic, butyric acid), lactic acid; further, numerous pigments, an albuminate containing iron, and in general many iron compounds, sometimes indeed free iron oxide (*Nasse*). *Malassez* and *Picard* have proved that the spleen contains more iron than its quantity of blood corresponds to.

The investigations of *Heidenhain*, published in 1875, concerning the presence and formation of zymogen in the pancreas, show that it accumulates in the pancreas cells quite independently of the spleen.

Very recently *Herzen* has communicated experiments on digestion, from which he draws the conclusion that the physiological condition for the transformation of zymogen into pancreatin is the formation of the splenic ferment assumed by *Schiff*, which ferment is dependent on the functional dilatation of the spleen. He considers, however, that the spleen does not enlarge with every digestion, and that when it remains small and anæmic, in spite of digestion, the pancreas digests no albumen.

Schiff, in his paper before the fifth International Medical Congress, held in Geneva, in 1877, reaffirmed that while peptogenous matters were drawn from the stomach, and the stomach unloaded itself, that the spleen at the same time elaborated a ferment which, going from the circulating blood into the pancreas, transformed a substance, probably albuminous, found in it, into pancreas-pepsin or trypsin, which latter energetically digested albumen, so that after extirpation of the spleen, the pancreatic juice lost its influence on the digestion of albumen wholly and permanently, while its other digestive properties were retained.

To test the accuracy of this assertion, *Ewald* (Einfluss der Milz auf die Pancreas-Verdauung. *Archiv für Physiologie*, II., S. 537) extirpated the spleen in a dog, and six days afterwards made a pancreatic fistula. The animal was in the act of digesting, and in three hours somewhat over twenty cubic centimetres of juice was collected, of a thin character and faint alkaline reaction. The juice digested fibrine and coagulated egg-albumen, changed starch into dextrine and sugar and emulsified fat, had therefore all the characteristics of normal pancreatic secretion. The spleen, therefore, is without influence on the pancreas.

Further, according to *Schiff*, extirpation of the spleen has no lasting influence on the absolute or relative quantity of red or white blood-corpuscles, and the enlargements of lymphatic glands, which exceptionally occur after removal of the spleen, come from a partial peritonitis.

Numerous experiments have been performed with a view of explaining the conditions under which changes in the size of the spleen occurred. Attention was chiefly directed to finding out through what nervous influence the contraction of the spleen was brought about, and whether it affected the morphological elements of the blood in the splenic vein. These questions were so much the more prompted since the presence of muscular fibres has been determined in the capsule and trabecular framework of the spleen. *Rudolph Wagner* first observed distinct contractions of the spleen in dogs in the year 1849. *Henle* observed them in the body of a person executed, which came to examination thirty-five minutes after death, in the year 1852 (*Henle* u. *Pfeiffer's Zeitschr.*, N. F. [1852] Bd. II., Heft 3, S. 299). In the year 1854, *Sinistra* (Commentat. physiolog. de funct. lienis. Groning, 1854) recorded contractions in the spleen of dogs and cats, which he produced by means of a rotation-apparatus. *Fick* (Zur Mechanik der Blutbewegung. Reichert und Du Bois' *Archiv* 1859, Heft 1, S. 8–13) in 1859 considered the contractility of the capsule and trabeculæ of the spleen of domestic animals as a fact. He observed in a full-grown ram that the spleen, which at first presented a flat surface, began to contract slowly under the action of atmospheric air. In 1863 *Tomsa* (Die Lymphwege der Milz. Sitzungsbericht der Wiener Academie, 1863, Bd. XLVIII., Heft 5, Abth. II., S. 652–666) gave a scheme of the contraction of the spleen. He described regular rows of flat muscular fibres running parallel to the veins, to which the ends of the trabeculæ, also containing flat muscular cells, were attached, at an acute or right angle. The contractions of the former, according to *Tomsa*, shorten the veins, the contractions of the latter distend them and keep them from collapsing during the contraction of the spleen. This arrangement is said to favor the flow of blood from the organ. In the year 1869, *W. Müller* expressed the opinion that the simultaneous contraction of the muscular fibres of the capsule and of the trabeculæ exercised a pressure on the parenchyma, as the result of which those parts of the latter, capable of motion, were forced to flow out. In order to obtain further points for the etiology of leucæmia, especially the nervous influence thereby, *Mosler* performed in the year 1869 the experiments described in his monograph on leucæmia, pp. 130–139. As confirmatory of the statements of *A. Bernard*, he found, when the electrodes of the induced current were brought in connection with the upper and lower ends of the spleen of a dog, after removal from the abdomen, that there was a diminution in length, and when the current went transversely through the spleen, there was a diminution in breadth of the organ. He could also obtain contractions of the spleen by means of the constant current. Electric irritation had a similar effect on passive congestion of the spleen, brought about by ligature of the splenic vein. A comparative examination of the blood from the splenic vein, made before and after application of the electric current, did not show a greater flow of white blood-corpuscles as the result of the electric irritation of the surface of the spleen.

Bulgak (*Centralbl. f. d. med. Wiss.*, 1876, 33, u. Virchow's *Archiv*, 69 Bd., S. 161) has since published further investigations concerning contraction and innervation of the spleen. He found that the quantity of white blood-corpuscles issuing from the splenic vein reached its maximum in the ordinary uncontracted condition of the spleen; that, on the other hand, this quantity decreased on contraction of the organ, and reached its minimum during the swelling of the organ; but when the spleen, previously swollen from any cause—for example, from ligation of the splenic vein—is made to contract, this quantity increases considerably.

While by curare and protracted narcosis the spleen loses its power of contraction, injection of quinine into the vein causes contraction of the spleen and increase of the white blood-corpuscles in the blood of its veins. Irritation of the semilunar ganglion produced a general and extreme contraction of the spleen in dogs and rabbits. In the upper part of the medulla spinalis, between the first and fourth cervical vertebræ, lies a mass of ganglion cells which governs the contractions of the spleen, *the reflex and motor centre of the spleen;* from it go centrifugal and centripetal fibres, which can be recognized as such in the nerves which surround the splenic vessels.

Through these experiments the nervous influence which, in medical practice, in the origin of splenic diseases, frequently does not appear distinctly, is placed in a clear light.

SPLENOTOMY.

BIBLIOGRAPHY.

BARRAULT, ERNEST: Etude sur la valeur de la splénotomie. Virchow's Jahresbericht, 1876, Band II., S. 426.—SHATTUCK, F. C.: Floating Spleen. Boston Med. and Surg. Jour., 1878, Aug. 22d.—ARNISON, W. C.: Brit. Med. Jour., 1878, II., 723–724.—NEDOPIL, M.: Die Laparotomie. Wiener med. Wochenschr., 1879, XXIX., 221–226.—POUCEL: Considérations sur la gastrotomie en général et particulièrement sur l'opportunité de la splénotomie. Marseille méd., 1879, XVI., 70–86.—ROGERS, H. W.: Buffalo Med. and Surg. Journ., 1878–9, XVIII., 367–369. —CZERNY, V.: Wiener med. Wochenschr., 1879, XXIX, 333–336, 367, 395, 457.

Barrault is of the opinion that extirpation of the hypertrophied spleen must be regarded as an admissible operation only under very exceptional circumstances. The indispensable conditions of it are, according to him, a healthy character of all other organs; also sympathetic symptoms of splenic hypertrophy threatening life, especially intolerable attacks of pain, moderate enlargement of the organ, and absence of adhesion to other organs or to the abdominal wall. The most important functions of the body and the general health appear to suffer no essential disturbances from extirpation of the spleen.

Shattuck collected twenty cases of splenotomy; *Nedopil,* twenty-three, and *Czerny,* twenty-five. Up to the time when the latter wrote, six cures had been reported. In all these, great nervousness was (they were all female patients) a prominent symptom. Other constant anomalies were not proved; even the changes in the blood were transient, and the enlargement of the lymphatic glands appears to be neither constant nor lasting.

CHRONIC SPLENIC TUMORS. PSEUDO-LEUCÆMIA.

In respect to its occurrence and etiology, pseudoleucæmia shows no essential difference from leucæmia. The greater number of those affected are of the male sex. Of thirty-three cases collected by *Richard Schulz* in which the lymph-glands formed the starting-point of the disease, there were twenty-four males and nine females. The age was given in thirty two patients; the greatest disposition to disease existed in childhood; it then diminished to the fortieth year. About this time a certain immunity appeared to occur, and from the fifty-fifth year on there was again a great disposition to it. In *Mosler's* clinic there have been seven cases treated, of whom five were men and two women.

The cases of disease were distributed over the different classes and callings. In age the patients were between one and sixty-nine years. All the cases observed by *Mosler* occurred in the middle period of life (his seven cases were 22, 33, 33, 35, 39, 43, 53 years old).

The beginning of the disease is often obscure, and no definite reason can be given for its occurrence. Some affected persons have not undergone any serious sickness, but, on the contrary, have always been healthy; many are even strikingly robust and strong. In other cases disease has preceded and circumstances have existed which may be placed in connection with the pseudoleucæmia.

In four cases intermittent fever preceded, sometimes immediately, sometimes years before (*Bonfils, Cossy, Cohnheim, Wunderlich,* 1866) in three cases the use of alcohol was proved (*Wunderlich,* 1866, *Lendet, Richard Schulz*). In two cases the disease followed convalescence from whooping-cough in children (*Hillier, Murchison*); in two cases it came after great emotions, once in a woman, after the death of her husband, the other time in a young man with mental depression following an unhappy marriage (*Hérard, Wilks*). In a few cases *Trousseau* assigns inflammatory diseases in the region of the lymph-glands first affected as the cause (otorrhœa, dacryocystitis, coryza chronica). In one patient distinct scrofulous affections were present and another suffered from syphilis. Once the disease followed twelve months after child-birth (*Barwell* and *Williams*).

The cases observed by *Mosler* were distinctly traceable to previous diseases, namely, diphtheritis, typhoid fever, and lepra.

PATHOLOGICAL ANATOMY.

The dead bodies usually show great emaciation, general anæmia, pale gray skin, flabby thin muscles; frequently there is general œdema, or at least œdema of the legs, scrotum, serous effusion into the peritoneum, pleura, pericardium; further, œdema of the lungs and of the brain.

The various lymphatic organs are affected in different succession, according as the splenic or lymphatic form of pseudoleucæmia is present.

Besides *Virchow* we are indebted to *Langhans* and *R. Schulz* for the accurate description of their anatomical relations, and their work forms the basis of the following description:—

The spleen is considerably, often enormously enlarged, sometimes soft, sometimes firm, at other times only hypertrophied, or strewn with numerous white, grayish-yellow, soft nodules as large as a pea or hazelnut, or larger, and which sometimes protrude above the surface of the section, sometimes do not, and are recognized as greatly enlarged Malpighian bodies. Their shape is normal, or it may be changed by mutual pressure, so as to be polyhædral or, under the capsule, conical. Their white or yellowish-white color, and the strongly contrasting brown pulp, give the surface of the section a very characteristic, strikingly variegated appearance. It is rarely that only a few, usually the majority of the follicles are diseased; also in spleens apparently only hypertrophic, the Malpighian bodies are often considerably enlarged, but they do not contrast with the surrounding tissue, because they have the same color. The entire volume of the spleen is enlarged in different degrees, according to the size of the follicles; the weight may reach two to three pounds. The capsule is often changed, thickened, and attached to the surrounding organs by adhesion.

Virchow considers that these nodules come from the pulp-tissue, while *Langhans* holds, with *Wilks*, *Billroth*, and most other authors, that they are changed follicles because their position and shape indicate this as well as their relation to the arteries; for they are located just as the follicles are in the adventitia of the latter, either clinging to the side or surrounding it like a sheath. The smallest nodules can be directly traced to the follicles, and it can only be a question whether the larger ones arise from one follicle or from the junction of several. *Wunderlich* considers that the latter is the case; yet it is not probable that all the larger nodules arise in this way. *Langhans*, in one case, because of their straight boundary line, considered most of them as enlarged follicles; *Virchow* and *Lambl* mention still larger solitary nodules, with a border not sharply marked at the periphery, but running over into the splenic pulp. Recently, *R. Schulz* has found that the larger nodules, in many cases, lose themselves in the surrounding tissue in radiating projections; in others they are encapsulated, and the capsule has been proved, microscopically, to be very compressed splenic tissue, which lies in concentric lines around the knots, so that great congestion is often present in this splenic tissue, the pulp veins are dilated, and in most cases numerous amorphous granules of hæmatoidine are heaped up around the nodules. *Schulz* does not regard these nodules found in the spleen secondary to lymphosarcoma or desmoid carcinoma, as metastatic like epithelial cancer, in which germs are carried away by the lymphatic system of vessels or blood, or, as in malignant sarcoma, in which metastases occur more especially through the veins, but they must be regarded as the result of a virus spread through the whole body, and causing an irritation, which becomes active here only later, when the new growth commences and forms in other organs also, as in the lymphatic glands.

Of the lymphatic glands, those of the throat are the ones which most frequently become diseased, then the retroperitoneal glands, the inguinal, mediastinal, and bronchial glands; afterwards come the mesentery, the lumbar and axillary glands. Much more rarely the cubital, those of the ham and the occipital glands, become affected, in conjunction with which usually the glands of the arm along the biceps muscle are also affected. (Of the thirty-eight cases collected by *R. Schulz*, the lymphatic glands of the throat were affected twenty-eight times, the retroperitoneal glands twenty-one times, the inguinal glands twenty times, the mediastinal glands eighteen times, the bronchial glands fourteen times, the mesenteric fourteen times, the lumbar glands twelve times, the axillary nine times, the cubital four times, those of the ham only once, and the occipital glands once.)

The lymphatic glands of the throat form tumors, the size of a dove's egg and larger, on both sides of the throat, which send prolongations down into the thorax, and in many cases compress the trachea. They stand in connection with the bronchial and mesenteric glands, which are also swollen, and which often form tumors the size of the fist, or even larger, frequently pushing aside and compressing the lungs, heart, and bronchi. If the axillary glands are swollen, they also form tumors of different sizes, which sometimes run in a chain towards the pectoral muscles, or along the biceps down the arm. The inguinal glands also frequently attain considerable size.

The skin over the outer glands is usually movable, and only very rarely adherent to the gland. The tumors are readily movable, feel elastic, soft, sometimes so fluctuating as to give the impression of a cold abscess.

From the mediastinal glands the tumors continue their course along the vessels to the abdomen and frequently form in front of the vertebræ colossal tumors three or four times the size of the fist, at times compressing the iliac veins, ureters, and so forth. The mesenteric glands form smaller tumors, the size of a bean or hazel-nut, which are scattered regularly through the mesentery. Sometimes also the lymphatic glands at the hilus of the spleen and in the transverse fissure are diseased, so that the gall-ducts are compressed and icterus produced.

On section, the smaller glands still reveal the normal structure, while the larger ones show a homogeneous, whitish-yellow, grayish-red surface and no further trace of normal structure. A cream-like, milky fluid can be scraped from the surface of the section, or it runs of itself from most tumors. There is no difference seen between follicular and medullary substance, the connective tissue septa which radiate into the interior of the lymph-gland from the capsule have disappeared and the capsule is frequently affected in the same way, and is seldom thickened by periadenitis. Sometimes it is broken through and the new growth increases in the fat tissue surrounding the gland or lying in the hilus. The new growth itself consists of a delicate, cobweb-like network, in whose meshes cells are imbedded, containing one or several nuclei.

The *liver*, which in thirty-eight cases was affected sixteen times, nine times with scattered nodules, is usually moderately enlarged in all dimensions, tolerably dense, has a smooth surface; and the acini appear to be somewhat large when the organ is diffusely diseased. If it is scattered with nodules, some of them are usually seen on the surface. On section, soft yellowish-white nodules as large as peas, beans, or hazel-nuts become prominent on the surface. They always lie in the region of the portal vein, never in that of the hepatic vein. They are seldom exactly inclosed in a capsule, but rather merge into the surrounding tissue. If the new growth is diffusely spread in the liver, great resistance is felt on cutting, and all the acini on the surface of the section are surrounded by light-gray borders, consisting of the mass of new growth. The surrounded hepatic tissue appears to be normal. The new growth begins in the outer layers of the adventitia of the vessels and is brought in connection with the perivascular lymphatic vessels running here.

In the *kidneys*, which, according to *R. Schulz*, become diseased in about a third of the cases, the nodules are sometimes in the cortical substance, sometimes in the medullary substance, and at times on the border between the two; the nodules are not completely encapsulated, but are gradually lost in the surrounding tissue. The kidneys are not very much enlarged, and are sometimes somewhat anæmic. Microscopically, besides an abundance of blood-pigment around the nodules, they are strewn with the remains of urinary tubules, with swollen and cloudy, nearly degenerated epithelium. The growth increases far into the interstitial tissue, between the tubules, whose epithelium also undergoes a fatty degeneration.

It is relatively seldom that the new growth attacks the *lungs*.

Sometimes, rarely, to be sure, the mucous membrane of the *digestive tract*, especially the follicles and Peyer's patches, are affected by the new growth. The mucous membrane is then swollen, relaxed, and diffusely infiltrated, sometimes having lentil-sized ulcerations with protruding edges. The follicles are swollen, prominent on the surface and on microscopic examination show the same new-formed tissue as in the lymphatic glands. The new growth appears to press forth from the vessels of the submucosa. The parotids, tonsils, and glands of the tongue are still more rarely affected; likewise the testicles.

The *heart* is healthy in most cases; sometimes it is a little flabby and soft, filled with many clots. A few times it showed extreme fatty degeneration; in two cases it was studded with nodules of the new growth. Sometimes diaphragmatic and perhaps omental nodules are present. The *medulla of bones* in the cases examined by *Schulz* contained peculiar gelatinous, gray, yellowish-red foci.

The *blood*, which is only in small quantity, is very fluid, not tending to coagulate, and readily deposits fatty precipitates. Accurate analyses are wanting at the present time.

SYMPTOMATOLOGY.

In *Mosler's* accurately observed cases, the relation of the disease to the lymphatic organs was a manifold one. In one case only the lymphatic glands were diseased and an enlargement of the spleen was not demonstrable. In the case following diphtheritis, lymph-glands and spleen were affected at the same time, indeed, the former so extensively that besides all the glands, the liver, kidneys, mucous membrane of the intestinal canal, as well of the larynx and urinary passages, shared in the lymphatic growth. In three other cases, where spleen and glands were affected at the same time, the former was chiefly affected, and in two cases the spleen only was diseased, without participation on the part of the lymphatic glands. *Mosler* therefore considers it practicable to distinguish a splenic and a lymphatic form of pseudoleucæmia, so that we may thus designate the starting-point of the affection. Whether we are justified in admitting a *medullary* form in addition, further investigations must decide.

The commmencement of the disease can be most definitely established when the external lymphatic glands or all of them swell together. Sometimes these remain free and the internal ones only swell, their enlargement usually being discovered later, in the case of the retroperitoneal glands, by feeling an abdominal tumor.

Of the superficial groups of glands, the lymphatic glands of the neck usually swell first (thirteen times in thirty-seven cases recorded by *R. Schulz*), less frequently the inguinal glands are first attacked (three times), still more rarely the axillary glands (once). The swelling then gradually goes over to the neighboring groups of glands, following the stream of lymph, so that, for example, from the glands of the throat a chain of tumors is formed towards the mediastinum. Sometimes the groups of glands enlarge in quite an irregular order, so that there is a slipping from one side to another, from the right side of the throat to the left axillary glands, the inguinal glands of the left side, and so forth. The patients in most cases are still relatively well.

Frequently little that is certain can be learned about the spleen, since its increase is less noticeable to the patients. A considerable enlargement appears never to have developed in so short a time as has several times been observed in the lymph glands. In all the cases observed by *Mosler*, the splenic tumor had already obtained a considerable size when he first saw them. The largest splenic tumors which he has yet seen occurred in connection with leucæmia and not pseudoleucæmia. This may be quite accidental, since, as the following notice shows, the increase in volume may be very considerable also in the latter disease.

In one of *Mosler's* cases of pseudoleucæmia, the left hypochondrium was very prominent, the intercostal spaces expanded, the contour of the spleen very distinctly palpable; when lying on the back the point of the spleen reached at the level of the tenth rib thirteen centimetres in front of the axillary line; from above downwards the splenic dulness measured twenty-three centimetres in the middle axillary line. The surface of the spleen was smooth. Notches could be distinctly felt in the edges of the spleen. Sometimes friction was to be felt over the splenic tumor. The patient complained of distention of the abdomen and of constipation, but respiration was not impeded by the splenic tumor. On the other hand, feeling of pressure and pain in the abdomen were experienced, which occurred mostly in the afternoon from four to six o'clock, constant weakness, tearing in the region of the sacrum, difficulty of moving the legs. Swelling of the glands could not be found. Anæmia was extreme; nose-bleed frequent. A microscopic examination of the blood showed no increase of the white blood-corpuscles.

Mosler has observed increase and diminution of the splenic tumor, both spontaneously and in connection with the use of drugs. Lasting pain has not been noticed either in the spleen or lymphatic glands. An unpleasant sensation of pressure and fulness was often observable in the left hypochondrium. In a series of cases, the occurrence of tumors was accompanied by febrile symptoms. Many times, together with increase of volume of the organs affected, other sorts of disturbances, namely, constitutional, occurred; in most of *Mosler's* cases there was hemorrhage. In other patients, enlargement of the spleen and lympathic glands exist for a longer or shorter time before other symptoms of disease are added.

The disturbance of nutrition is characterized by extreme pallor of the mucous membranes, by a waxlike greenish appearance of the skin, by great emaciation dependent partly on disappearance of the panniculus adiposus, partly on the shrinking of the muscles. During the use of large doses of quinine for twenty days, *Mosler* observed a marked increase of weight in one case, and also marked diminution of the muscular weakness. Many patients show great nervous irritability which manifests itself frequently by increased frequency of the pulse, the temperature remaining normal. In a patient who suffered at the same time with amenorrhœa and spinal irritation, nervous palpitation of the heart was very marked. In women, cessation or irregularity of the menses is the rule. The disturbed formation of blood is shown by another series of symptoms. *As a result of their poor nutrition the vessels have a tendency to rupture.* *Mosler* saw frequent profuse nose-bleed in four of his cases, twice melæna, and once bleeding from the gum. Of eighteen cases *Müller* saw petechiæ of the skin in four. Though disturbances of circulation caused by the white blood-corpuscles do not exist in pseudoleucæmia, yet in most cases general or local œdema of the body occurs, caused partly by the hydræmic character of the blood, partly through pressure of the tumors on the larger veins of the body; serous effusion into the pleura or peritoneum may occur, the latter, perhaps, dependent on disease of the liver. Serous effusion into the pericardium sometimes occurs. Usually the symptoms of cachexia and marasmus can be established in a great number of organs. The extreme dyspnœa which patients suffer in advanced stages is caused partly by the swollen bronchial and mesenteric glands, partly by the hydrothorax present, and also by the pushing up of the diaphragm resulting from ascites combined with the hepatic and splenic tumors. It is probable also that a part of the dyspnœa depends on the changed character of the blood, the diminution of the red blood-corpuscles. Many patients have a marked bronchitis, others short, often spasmodic cough without expectoration, depending on the pressure of the tumors on the recurrent nerve. The appetite is often much impaired, patients have a bad taste in the mouth, sometimes fetor ex ore. In consequence of pressure of the splenic tumor on the intestines, obstinate constipation is added. Disturbance of the stomach is brought about by the same cause in some cases, with foul belching and vomiting. Towards the end of the disease, with increasing exhaustion, diarrhœa is added after protracted constipation has preceded.

The function of the kidneys is performed differently, according to the stage of the disease; in the commencement the quantity of urine being normal, in advanced marasmus with dropsy, very much diminished. Then with a high specific gravity and acid reaction, there is an abundant sediment of urates.

During a period of experimentation, lasting thirty-five days, a patient in *Mosler's* clinic, with splenic pseudoleucæmia, received a weighed quantity of food (every twenty-four hours two quarts of milk, four soft boiled eggs, a quarter of a pound of meat, a pound of bread, a bottle of beer). The amount of urine for the first fifteen days averaged 1420 cubic centimetres: its specific gravity 1022, urea 38.135, uric acid 0.18, chloride of sodium 9.021 grammes. During the following ten days the patient received the same weighed diet, and in addition one gramme of sulphate of quinine every twenty-four hours, and the following average was obtained : amount of urine 1469 cc., specific gravity 1020, urea 35.677, uric acid 0.395, chloride of sodium 6.17. When the patient, under the same conditions, during the next ten days took two grammes of sulphate of quinine, the average quantity of urine was 1500 cc., specific gravity 1020, urea 38.299, uric acid 0.374, chloride of sodium 5.616.

The considerable secretion of urea is striking. *It was not changed* *by quinine; on the other hand, the secretion of uric acid was increased, that of the chloride of sodium was diminished.*

Strümpell has also recently proved that in anæmia splenica there is a considerably increased destruction of albumen in the body, and Dr. *Schneider* has also proved several times in progressive pernicious anæmia that there was an increase of the urea in the urine. It is not impossible that further investigations may render feasible a nearer fathoming of the diseases belonging here and hitherto grouped together elsewhere.

Sometimes the urine contained albumen dependent either on passive congestion of the kidneys, or on the new growths developed in them. Biliary coloring matter is found in complication with icterus which arises in rare cases from pressure of the tumors on the gall-ducts.

DIAGNOSIS.

Under the name pseudoleucæmia we have included those cases of disease which depend on real new-formation of spleen or lymphatic glands, splenic tumor and glandular enlargement, *without accompanying leucæmic character of the blood*, but characterized on the other hand by a greater or less pronounced tendency to formation of lymphatic glands *de novo* in other organs. We must distinguish a splenic and lymphatic form of pseudoleucæmia. The most frequent occurrence is the combination of both forms. On account of the superficial position of the lymphatic glands, we can recognize pseudoleucæmia lymphatica more accurately and earlier, and therefore our knowledge of it has been essentially advanced in the last few years. It has been called glandular sarcoma and lymphosarcoma by surgeons and pathologists, and in addition to the general or multiple form which we are considering, they also distinguish a local circumscribed form. Most cases belong to this latter form. They are of a local nature, affect only one or a few groups of glands lying directly behind each other in the direction of the lymph-stream, as of the lower jaw, the axilla, mediastinum, and so forth. Their reaction on the blood and general nutrition is not great, on account of their limited extent. Their anatomical significance lies in a simple hyperplasia, sometimes with preponderance of the connective tissue reticulum, so that two conditions are to be distinguished—the *pure hyperplastic* form with new growth of follicles and lymph-channels, better known through the investigations of *D. Müller* (*Zeitschrift für rationelle Medicin*, 3, R. 20, 129, 1863), and the *indurative* form. The difference between the two, as *Billroth* has shown (*Beiträge zur path. Histologie*, S. 168, 1858, und *Virchow's Arch.*, XXI., S. 439, 1861), is that in the former the microscopic elements maintain the same relative relation and the same arrangement as in the normal gland, while in the indurative form trabeculæ and capsule are transformed into a tissue resembling the follicular substance through infiltration with lymphatic cells, so that the structure of the gland is perfectly homogeneous.

From an anatomical stand-point this should be designated as simple or indurative hyperplasia, and the name lymphosarcoma should be considered applicable only when many groups of glands are affected and the affection assumes a decidedly malignant character. It is malignant as well through reaction on the whole nutrition of the body as through the occurrence of new growths of similar structure in other organs. In this the affection bears a great resemblance to leucæmia, and therefore clinically the name pseudoleucæmia is fitting. The clinical diagnosis in some cases of pseudoleucæmia is rendered difficult in that the new growths in the rest of the organs are sometimes absent and sometimes present, so that *Langhans* has distinguished (*Virchow's Archiv,* 54. Bd., S. 510) a form only affecting the gland and a metastatic form. The first form is probably very rare and its occurrence is by no means certain, since the medulla of the bones has not been duly examined.

Langhans, following *Virchow,* has distinguished a hard and soft variety of metastatic lymphosarcoma, which, though in their extreme forms very different, yet in many cases merge into each other. Thus in the same case the glands may be of different consistency; for instance, the small ones hard, the large ones softer; or the metastatic deposits of the hard variety may be more soft and juicy. A separation, therefore, on this principle of the cases scattered through literature will always be somewhat arbitrary (*Langhans*).

The soft variety more nearly resembles leucæmia, and is distinguished from it only through the normal proportion of white blood-corpuscles. The resemblance includes the clinical appearances as well as the results of autopsy, the gross and microscopic structure of the primary tumors as well as the secondary deposits.

Langhans has definitely determined the character of the hard variety of lymphosarcoma; the lymphatic glands always form the commencing point of the affection, and are then dense, stiff, fibrous, almost fibro-cartilaginous in consistency. In many cases, this characteristic is so pronounced that it is sufficient alone to establish the diagnosis. Other cases exhibit a gradual change in the consistency. In hard sarcoma there has hitherto been a pure lymphatic and a mixed lymphatico-splenic form recognized. A primary disease of the spleen has not yet been proved; for this reason the hard form does not have quite the same significance for our consideration as the soft lymphosarcoma which has been admitted to occur primarily in the spleen. *The name pseudoleucæmia is alike applicable, however, to the soft and hard forms, since the clinical symptoms are almost identical in the two* (*Langhans*).

Unfortunately, up to the present time we are still unable to determine precisely the nature and origin of pseudoleucæmia, either in respect to its different forms or in relation to other processes, as we are still in complete ignorance of physiological as well as of pathological processes in the blood-making organs, and do not know the relation of the latter to each other. When the history does not give us information in combined lymphatic and splenic pseudoleucæmia in an advanced stage, we cannot accurately determine whether the spleen or the lymphatic glands have been the starting-point of the affection. The anæmia accompanying this process can neither be distinguished from other forms of general anæmia, nor can the anæmias of splenic and lymphatic pseudoleucæmia be accurately distinguished from each other. On account of ignorance in this direction, we are often unable to say of a splenic tumor which has been ascertained by clinical examination, whether we have to do with pure hyperplasia of the spleen, with splenic pseudoleucæmia or commencing leucæmia. In such cases, continued observation only with further development of the process can establish the diagnosis. A repeated examination of the blood is the only criterion for the differential diagnosis of pseudoleucæmia and commencing leucæmia.

We cannot say whence the striking difference in the character of the blood in these two affections comes. The cause should be sought in a difference in the anatomical change of the lymphatic glands, and just here there are decided loop-holes in our knowledge, as we know nothing of such differences. The fate of the lymph-channels has not been accurately investigated, either in leucæmia or pseudoleucæmia.

There are scarcely two diseases which are so similar to each other as pseudoleucæmia and true leucæmia. It is easily explained, therefore, how the opinion was entertained that pseudoleucæmia was a preliminary step to leucæmia; it represented the latter disease in an earlier period before a pronounced leucæmic character of blood could arise. Cases running a very rapid course, like the one observed by *Cohnheim*, and one described by *Trousseau*, the former lasting scarcely three months, the latter ending fatally through suffocation before cachexia had occurred, might support the assumption that the short duration of the disease had not allowed a marked leucæmic change in the blood to occur. *Mosler* has seen the most extreme degree of marasmus occur after long continuance of the disease, without observing any change in the character of the blood.

The difficulty of diagnosis is increased, in that there are many transition forms between leucæmia, pseudoleucæmia, and pernicious anæmia, which show moderate increase of the white, and changed character of the red blood-corpuscles.

Mosler described, in his monograph on leucæmia (p. 220), the case of a splenic tumor coming on after pernicious intermittent fever with great anæmia and hæmorrhagic diathesis, where the white blood-corpuscles were not increased; on the other hand, many red blood-corpuscles had tail-like processes. Many were changed to the biscuit-like forms described by *Friedreich*. He made a diagnosis of extreme anæmia resulting from a splenic tumor, anæmia splenica, and explained the condition of the blood thus, that they were not finished red blood-corpuscles, but that they were still transitional between red and white blood-corpuscles, so that probably it was the initial stage of leucæmia. He had the patient under observation a long time, and the splenic tumor and character of the blood remained unchanged; it did not become leucæmia.

We do not yet know the deeper distinction between pernicious anæmia and splenic pseudoleucæmia; there are transition stages which are capable of a different explanation.

COURSE AND PROGNOSIS.

We must distinguish between cases running a very rapid course and those which show a slower development of symptoms. In the latter, a longer or shorter pause in the disease occurs. Drugs have a salutary influence. *Mosler* believes, with *Wunderlich*, that in the first stages a cure is possible. His experience is, that cases of disease beginning primarily in the lymphatic glands (pseudoleucæmia lymphatica) have a more rapid course and are less amenable to therapeutic measures than those which commence in the spleen (pseudoleucæmia lienalis). Among the former, those run the quickest course in which from the beginning a large number of glands are very quickly affected; they lead to death usually in the course of a year, many last one to two years, and only a small number over two years. They are, as a rule, those in which for weeks and months the glands first diseased remained only moderately enlarged, or in which the primary splenic tumor at first showed but few changes, and only later a general development of the glands supervened. The prognosis, therefore, in the cases first mentioned, is absolutely unfavorable; it is more favorable in those which show a less rapid increase of the disease.

THERAPEUTICS.

All drugs are inefficient in the above-mentioned cases of general and rapid development of the disease. It is only exceptionally that we can bring about an apparent pause by the exhibition of preparations of iron, quinine, or iodine. The favorable results of *Czerny* (*Wiener med. Wochenschrift*, No. 28, 1874) in cases of less rapid course deserve attention; he alternated between internal administration and parenchymatous injection of solutio arsenicalis Fowleri in malignant glandular lymphoma. Complete cure resulted within seven months in a patient who had taken seven hundred and forty-six drops internally, and seventy-six injections of ten drops each. The treatment of a woman in *Mosler's* clinic with a considerable splenic tumor (15:13) by simultaneous use of cinchona preparations and parenchymatous injections into the enlarged spleen, showed that it was also of service in pseudoleucæmia lienalis. The patient was ordered: Blaud's pills, Carlsbad salts, a subcutaneous injection twice daily of a solution of amorphous hydrochlorate of quinine (1 to 5). Immediately after every injection, ice-bags were applied to the spot where they were made, and allowed to remain several hours. It was due to this fact that only slight local inflammatory appearances were observed to follow. Moreover, great care was taken to insert the sharp canula as deeply into the subcutaneous tissue as possible. Within sixteen days they were able to make thirty-two injections into different portions of the body. A distinct diminution of the size of the spleen was then proved. Its front end had retreated three fingers' breadth behind the umbilicus, it reached only eight centimetres in front of the linea axillaris, its vertical extent was eleven centimetres.

As it was believed that a rapid diminution of the splenic tumor could not be attained by further use of quinine, it was determined to resort to parenchymatous injections. An ice-bladder was first applied to the splenic region for many hours, so as to lessen the amount of blood in the organ through contraction of the muscular tissue of the spleen. Then the anterior end of the spleen was grasped and the surface of the organ pressed as closely to the abdominal wall as possible. A very sharp subcutaneous syringe was pushed through the abdominal wall and a syringeful of a two-per-cent solution injected (about twenty-two drops). The splenic parenchyma was felt to present a certain resistance to the syringe. The patient also complained of severe pain during the injection, so that one centigramme of morphia was injected in the same spot immediately after, and during the next twenty-four hours ice-bags were applied to the spleen. The same procedure was frequently repeated at certain intervals, and in the last injections solutio arsenicalis (1:10) was used. A distinct diminution in the size of the tumor was determined after beginning the parenchymatous injections. The patient was discharged from the hospital in a very much better condition.

The admissibility of injections into the splenic parenchyma is established by this observation. *Mösler* has since repeated it several times. Certain precautions are to be observed in it. A long use of what we may term spleen medicines must precede, in order through contraction of the contractile elements of this organ to diminish the amount of blood in it. An ice-bag is to be applied to the splenic region for several hours just before the parenchymatous injection. Only those cases should be selected for parenchymatous injection in which the tumor lies closely applied to the abdominal wall, or in which it is possible, by seizing the anterior end of the organ, to bring it very near to the wall, and in those in which there is no hemorrhagic diathesis.

In another case of splenic pseudoleucæmia, *Mosler* obtained a very great diminution in the size of the tumor and improvement in the general condition by giving consecutive large doses of quinine. The patient, who was thirty-five years old, received daily for ten days one gramme of muriate of quinine, and in the following ten days one gramme twice a day. He bore the large doses without special discomfort. Later he used the cheaper chinoidin pills, and afterward oleum eucalypti in capsules.

Wunderlich observed improvement in one case through the internal use of iodide of potassium. As subsequent treatment, iron preparations are to be recommended.

Operative treatment is to be discountenanced in general, in the multiple form of gland tumors.

LEUCÆMIA.

BIBLIOGRAPHY.

Biesiadecki: Leukämische Tumoren der Haut und des Darmes mit einigen Bemerkungen über den leukämischen Process selbst. Oesterr. med. Jahrbücher, 1876, Heft 3.—Perls: Lehrbuch der allgem. Pathologie. Stuttgart, 1877, I., S. 373.—Cohnheim: Vorlesungen über allgemeine Pathologie. Berlin, 1877, I., S. 387.—Klebs: Archiv für experimen. Pathologie, Bd. IV., S. 110.—Robin: Leçons des humeurs, 1874, p. 267.—Mosler: Klinische Symptome und Therapie der medullaren Leukämie, Berlin, 1877.—Neumann: Ueber myelogene Leukämie. Berlin, 1878, A. Hirschwald.—Ponfick: Beiträge zur Lehre von der Leukämie. Virch. Arch., Bd. 67, 1876.—English: Zur Lehre der medullaren Leukämie. Wien, 1877, Czermak.—Kelsch: Arch. de phys. norm. et pathol., Nr. 3 u. 4 Virchow's Jahresber., 1875, Bd. 2.—Litten, Dr. M.: Berlin. klin. Wochenschr., 1877, Nr. 19.—Litten, Dr. M., u. Orth, J.: Berl. klin. Wochenschr., 1877, Nr. 51.—Küssner, B.: Berl. klin. Wochenschr., 1876, Nr. 9.—Huber: Deutsch. Archiv für klin. Medic., Bd. XIII.—Schepelern:—Virchow's Jahresber., 1873, II., S. 298. —Salomon: Reichert u. DuBois' Archiv, 1876, S. 762; Verhandlungen der Physiolog. Gesellsch. zu Berlin, 1877, Nr. 1.—Patrigeon, G.: Recherches sur le nombre des globules rouges et blancs, etc., Paris, 1877.—Zenker: Ueber die Charcôtschen Krystalle. Deutsches Archiv für klin. Med., XVIII., 1.—Longuet: Progrès méd., Nr. 32.—Ordenstein: Centralbl. für die med. Wissensch., Nr. 52.—Huber, K.: Archiv der Heilkunde, XVIII., S. 485.—Legg, Wickham: St. Barthol. Hosp. R., X. Virchow's Jahresber., 1876, Bd. I., S. 326.—Kretschy, Dr. F.: Wiener med. Wochenschr., 1878, Nr. 8.—Simmons, Dr. G. B.: Pacific Med. and Surg. Jour., Dec., 1877, 20 Bd. Wiener med. Wochenschr., 1878, Nr. 8.—Cabot, A. T.: Boston Med. and Surg. Jour., 1878, XCIX., 526–527.—Flint, A.: Med. and Surg. Reporter, Phila., 1878, XXXIX., 329–330.—Oeller: Beiträge zur pathologischen Anatomie des Auges bei Leukämie. Arch. f. Ophth., Berl., 1878, XXIV. (Abth. 3), 238–256.—Richardson, J. G.: Improved Method for Detecting L. During its Early Stages, etc. Tr. M. Soc. N. Y., 1878, 146–155.—Mosler: Ein Fall von primärer lymphatischer Leukämie. Arch. für path. Anat., etc., Berlin, 1879, LXXV., 543–547.—Bouchut: De la leucocythémie aiguë causée par la diphthérite. Gaz. d. hôp., Paris, 1879, LII., 153–155.—Gowers, W. R.: Splenic Leucocythæmia. Syst. Med. (Reynolds), London, 1879, V., 216–305.—Carpenter, A.: Leucocythæmia. British Med. Journ., London, 1879, I., 821.—Vulpian, A.: Leucocythémie splenique. Clin. méd. de l'hôp. de la Charité, Paris, 1879, 487-500.—Bousfield, E. C.: On a Hitherto Unnoted Feature of the Blood in L. Lancet, London, 1879, II., 8.—Welch: Lancet, London, 1879, II., 46.—Delrieu, M.: Quelques consid. clin. sur trois cas de l. Paris, 1879, pp. 42, 4to.—Mosler, F.: Zur Symptomatologie der medullären Leukæmie. Deutsches Arch. f. klin. Med., 1879, XXIV., 342.—Mosler, F.: Ueber hämorrhagische Diathese u. dadurch contraind. op. Eingriffe bei Leukämie, etc. Zeitschrift für klin. Medicin, Bd. I., Heft 2, 265, 1879.—Howard, R. P.: Montreal General Hospital Reports, 1880, Vol. I.—Peabody, Geo. L.: On Persistent Priapism, not Connected with Lesion of the Central Nervous System. N. Y. Med. Jour., May and Sept., 1880.—Gowers: Reynolds' System of Medicine, Leucocythæmia, 1879.

Since *Mosler's* monograph, published in 1872, the attention of investigators has been turned to the myelogenous form of leucæmia. It has been attempted to establish the relation of the different forms of leucæmia to each other, and, according to *Mosler*, we are justified in placing this with the other forms. The expression medullary leucæmia is recommended for general acceptance.

Really the idea that in leucæmia the blood-change was primary dates back to *Bennet*, who, as is well known, considered his first case as primary suppuration of the blood, an idea which *Virchow* refuted with great success. Also *Robin* (Leçons des humeurs, 1864, p. 267) and *Biesiadecki* have again advanced the opinion that the swelling of the spleen and lymphatic glands is not the exciting cause of the disease, but is the result of the leucæmia, which disease, according to *Biesiadecki*, arises from the white blood-corpuscles not undergoing the normal metamorphosis to red corpuscles in the blood-current. If we regard *Biesiadecki's* view as really correct, that the red blood-corpuscles spring from the white ones, it is very much more important to deduce an interference of this transformation leading to leucæmia from an alteration and deficient action of those organs on which the transformation is imposed, particularly the medulla of bones, than, as *Biesiadecki* does, to assume degenerative processes which the white corpuscles in the blood undergo, making them incapable of their physiological metamorphosis (*Neumann*).

Kottmann's argument that there is no lack of exhaustively investigated cases in which a considerable increase of the white corpuscles is found without the slightest change being proved in the blood-making organs (glands, medulla of bones, lymph-spaces) is weak, in that the observations cited by him as proof all belong to a time when no attention was paid to the medulla of bones at autopsies, and it does not follow, therefore, from lack of accounts of pathological changes in this organ that it was normal. Besides, in one of the cases mentioned by *Kottmann* (*Gubler*), a splenic tumor is specially mentioned in the account of the autopsy.

Certainly *Virchow's* doctrine of the secondary importance of the change in the blood compared to the pathological conditions of certain organs has received substantial support through the proof given by *Neumann* that the medulla of bone is to be reckoned as a blood-making organ, and that in leucæmia it frequently presents so striking a change. Although, in the present state of our knowledge of the physiology of the blood, we are unable directly to refute the opinion that leucæmia is a disease of the blood itself, independent of the organs of the body; this doctrine, at present at all events, is without any positive confirmation, while, on the other hand, the fact is established that thus far no case of leucæmia has been observed in which the autopsy did not show a disease of one or several of those organs which we are warranted in considering to have an influence on the composition of the blood. Every one conversant with the recent literature of leucæmia knows how very much opinions differ at the present time as to the position to be assigned to the medulla of bones in this regard, in distinction to the spleen and lymphatic glands.

Zenker (*Deutsches Archiv für klin. Med.*, Bd. 18, S. 134) a short time ago expressed the opinion that the spleen stood foremost in importance in leucæmia.

In the etiology of leucæmia, exposure to cold deserves to be mentioned. Of especial value are the statements of the ship's captain treated by *Mosler* for primary medullary leucæmia. Pain in the breast-bone came on after an exposure to extreme cold, which occurred in a four days' storm in the winter time off the coast of Sweden, followed also by a winter spent on his ship, which was frozen in where he could not keep warm, even in bed. *Mosler* mentions also a marked case of splenic leucæmia resulting from a severe cold.

For counting the number of the corpuscles, the method of either *Malassez*, *Hayem*, or *Gowers* is indispensable.

The symptomatology of medullary leucæmia is naturally less worked out than of the other forms. The first and only symptom is pain in the bones occurring spontaneously and on pressure. The most frequent seat of pain is the sternum. The pains in the bones have great significance in the cases of leucæmia in which they are observed, because, according to *Mosler's* observations thus far, medullary affection may be diagnosticated from them with certainty. Thickenings of bones occur as well as pain. Absence of swelling of the bones or absence of pain does not exclude leucæmic hyperplasia of the medulla of bones, which may develop or even reach a high degree without giving rise to visible changes on the surface of the bones. A change in the bones first appreciable externally will occur when the lymphoid infiltration has passed over to the periosteum, where the thickening may exhibit apparent fluctuation.

Mosler, in a case of medullary leucæmia, reported at the Leipzig Naturforscher-Versammlung, first proved that extreme sensitiveness of the sternum without thickening of the bone was a symptom of leucæmic hyperplasia of the medulla of the bone; on the other hand, there were no changes to be found in the long bones. He also reported in the *Berliner klin. Wochenschrift*, 1876, No. 49, a case of medullary splenic leucæmia, where the symptoms were much more pronounced than in the above case. Very soon after extreme exposure to cold, without any previous injury, pain was felt along the sternum, so severe that work had to be stopped. The pain was spontaneous, and greatly increased by pressure, especially by percussion. The sternum was more than usually prominent, and two circumscribed places could be distinguished on it where the sensation of elasticity was imparted to the finger. In other places, quite as painful on being handled, nothing was found. The heads of the ribs, especially the second to the sixth, were swollen and painful on pressure. Examination of the rest of the bones showed but little that was abnormal, only two places corresponding to the posterior superior spinous process of the left ilium and the left trochanter were painful on pressure and movement. By means of a Middeldorpf harpoon the softened sternum was explored during life, and a blood-stained mass was brought out by the canula, showing the same large white blood-corpuscles.

which were so striking in the blood. In two other cases of secondary medullary leucæmia observed since, there was pain in the sternum without prominence, also without changes of the rest of the skeleton. In a third case of splenic-medullary leucæmia, besides pain in the sternum and ribs, there was also prominence of the same. *Mosler* has called attention to the occurrence of small, circumscribed, soft tumors on the sternum, and *English* has observed thickenings of the long bones (on both femurs). Certain bones were thickened, others not. The symmetrical occurrence of such tumors of long bones is striking. Besides pain, the appearance of tumors of the bones with increase of white blood-corpuscles may be regarded as indicative of hyperplasia of the medulla, especially when variations occur in the tumors corresponding to variations in the general condition.

Biesiadecki recently observed the development of numerous tumors in the skin of the face, forehead, breast, and extremities. In the largest nodules, the medullary mass extended into the subcutaneous fat-tissue, and was sharply bounded from the neighboring tissue, while the boundary of the smaller tumors was indistinct. Histologically they quite resembled leucæmic nodules in other organs, also the enlargements of the spleen and lymphatic glands.

The *diagnosis* of *medullary* leucæmia presents special difficulties, as may be judged by what has preceded, since pain in the bones and enlargements of the same may be wanting, and since, moreover, they usually occur only in single places, and by no means equally distributed over the skeleton.

Whether a medullary affection may be diagnosticated from the presence of medulla cells in leucæmic blood, when symptoms connected with the bones are absent, only a large number of observations can determine. In *Mosler's* three cases of secondary medullary leucæmia, there were at the same time symptoms connected with the bones and medulla cells present in the blood, the former not very pronounced, the latter in smaller number than in his case of primary medullary leucæmia. He does not commit himself to the opinion that this is usual. The observations thus far made suggest that, in affections of the bones, not only the result of traumatism, but also in those arising from exposure to cold, a systematic examination of the blood be made to see if it may not be a first beginning of medullary leucæmia. The examination must not merely be confined to those cases in which a recent disease of the bones exists, but also those cases must be investigated in which there has ever been previous bone disease. Former inflammatory processes in the bones deserve especial attention in cases which have not gone on to suppuration, also concussion of bones, since there is no doubt that in both processes a severe irritation is exerted on the medulla and a transference of substances into the blood follows, as is seen from the high fever occurring in these diseases. If, after the termination of the process, no overfilling of the medulla of the bone with new-formed elements remains behind, any new irritation may cause increase of abnormal constituents in the blood (*English*).

We have some very interesting observations on the *duration* and *course* of *medullary* leucæmia, by which it appears that the course may be strikingly rapid or very protracted. *Litten* reports an extremely rapid case running its course in from four to five days, a fact which is more readily understood when we learn that the patient was a cachectic individual whose elaboration of blood was already much impaired (anæmia perniciosa). In the case described by *Küssner*, the patient, previously quite well, died after eighteen days of the disease, which first came on like an acute infective disease with a chill; and in *Immerman's* case death resulted in six weeks after the first symptoms. In both these cases, the disease ran its course with severe febrile typhoid symptoms, and, if no examination of the blood had been made during life, they would have been regarded probably as typhoid fever. In *Mosler's* case of primary medullary leucæmia, on the other hand, it was nine years after the commencement of the disease before the patient felt obliged to seek the aid of a physician, and under treatment he so far improved that, in April, 1878, he announced his intention of being married again. There appears to have been here a very long interval between the original disease of the bones and the dissemination to other lymphatic organs. It is said to have been six years after the affection of the sternum, resulting from cold, that the abnormal sensations occurred in the left hypochondrium, and when last reported, enlargements of the lymphatic glands were scarcely to be felt. The case reported by *English* appears to indicate a protracted, latent stage of medullary leucæmia. What factors aid in the production of a general disease can only be partially conjectured. Probably they are causes which exert a new irritation on the medulla of the bones, and increase its function, as traumatism, cold, etc. The varied rapidity in the course of medullary leucæmia makes the secondary affections of the spleen, lymphatic glands, liver, occur sometimes sooner, sometimes later. There is a similar comportment also with the secondary bone affections in primary splenic and lymphatic leucæmia. In one of *Mosler's* recent cases, pain in the sternum came on six months, and in another nine months after the splenic tumor had been diagnosticated. In its further course, the disease does not keep pace in the different organs, and an organ secondarily affected may make more rapid advances than the primary one, so that, after a time, deeper changes are met with in the former, and mistakes may very readily arise in regard to the order of the appearance of disease in the different organs.

THERAPEUTICS.

Wilson Fox, following *Broadbent*, gave a patient with splenic leucæmia at first one-fiftieth of a grain of phosphorus, later, one-thirtieth of a grain three times a day, with signal success. The strength increased, fever soon disappeared, the spleen became smaller, and the white blood-corpuscles (before increased twenty times) gradually diminished in number, so that, after about three months, the number was normal. *Moxon*, *Greenfield*, *Jenner* saw no result from this treatment. *Gowers* saw the number of the white blood-corpuscles diminish under the use of phosphorus, while *Goodheart* found no influence on the blood, but a general improvement. The discussion on these cases presented before the Clinical Society of London shows great doubt regarding the action of phosphorus.

Broadbent recapitulates the cases in which he had used phosphorus, according to which he claims to have seen favorable effects in one case of splenic leucæmia and in a few cases of lymphadenoma and essential anæmia, but he does not regard the drug as a specific in leucæmia. Of the other favorable cases mentioned, only that of *W. Fox* remains, and the real leucæmic nature of it is doubtful (*Riess*, *Virchow's Jahresber.*, 1877, II., 262). In December, 1877, *Mosler* gave a patient with lymphatico-splenic leucæmia, in whom enumeration of the corpuscles with Malassez's apparatus (a mean of five counts) gave 1,992,800 red and 175,360 white blood-corpuscles, 0.15 of phosphorus without any effect within twenty-five days; neither the lymphatic glands nor the spleen became smaller, fever continued, and the above proportion of white and red blood-corpuscles remained the same.

DaCosta made subcutaneons injection of ergotin in two cases of splenic leucæmia (five grains every second day) with good result, but *Mosler* did not obtain anything specially favorable with it.

Recently Dr. *Simons*, of Sacramento, undertook splenotomy in a man forty-three years old, suffering from splenie leucæmia. There were dense adhesions to the diaphragm, and the patient died of hemorrhage two hours and a half after the operation. This is fresh proof that splenotomy is contra-indicated in leucæmia, on account of the existence of an hemorrhagic diathesis.

Of the cases of leucæmia observed by *Mosler*, more than fifty per cent were complicated by hemorrhage.

Of the 13 who had hemorrhage, 2 were women, and 11 men, which bears out the observation that the male sex has a greater disposition to leucæmia than the female. With regard to age, in ten cases it was 35 to 45 years, in two children from 9 to 13, in one case in a young man of 19. The statistics show that hemorrhage may occur in all forms of leucæmia; neither any fixed age, sex, long or short duration of the disease, insures against hemorrhage. The nose is the most frequent seat of hemorrhage, though it may occur in any part of the body, either external or internal.

Bousfield reports the presence in the blood of irregular granular masses of considerable size, protoplasmic in their nature (as shown from the fact of their exhibiting slight amœboid movements), and somewhat cylindrical in shape. They are not found in every case.

A very good illustration of the danger from hemorrhage is the following case: In the summer and autumn of 1877, *Mosler* had under treatment a patient from Finland, with advanced splenic leucæmia. As a last resort, wearied out with his disease, he wished to try splenotomy. *Péan* to whom he had written and *Mosler* both advised against the operation.

The hemorrhagic diathesis, which had previously been only surmised, was shown by a profuse hemorrhage following the artificial opening of a perineal abscess. All styptics were ineffective and digital compression had to be applied for many hours before the hemorrhage ceased. Death resulted from spontaneous intra-abdominal hemorrhage in the middle of January, 1878. *Mosler* also reports the favorable influence of quinine with Schwalbach water in a case of lymphatic leucæmia; three to six goblets were taken daily; the tumors of the glands became smaller, the suffering from stomatitis leucæmica became less, and the general condition much better. By the use of the iron baths of Rooneby, in Sweden, he observed a retrogression of the medullary affection in the case of a patient from Finland. Pain in the sternum subsequently disappeared, pain or swelling of other bones did not occur. No improvement of the leucæmic cachexia was obtained, however. In one cubic centimetre of blood examined with the Malassez counting apparatus, there were 2,105,600 red, and 451,200 white blood-corpuscles (5 to 1); on July 16th and on September 12th, after returning from Rooneby, there were 2,018,400 red, and 433,200 white blood-corpuscles (5 to 1).

The *treatment* of medullary leucæmia appears to have more favorable prospects than the other forms. As we regard the marrow of bones to be the starting-point of the disease, we must see that all irritations which act on the marrow, as injury, concussions, the action of cold, and others shall be kept away. As long as the disease of the bones is localized, we must treat it energetically. When the disease is further developed, both local and general treatment must be adopted. Of the former, the constant application of cold (ice-bag) has done good service (*English*). In addition, the parenchymatous injection of solutions of carbolic acid of various strengths is to be used. *English* has used iodide of potassium internally 1.5 to 2 grammes in a day with such good effect that he has no reason at present to change his treatment. *Mosler* obtained favorable results from the pill of quinine, ol. eucalypti, and piperine, already mentioned. Not only did the affection of the bones diminish (*Berlin. klin. Woch.*, 1876, No. 49) and the splenic tumor become decidedly smaller, but also the number of white corpuscles in the blood greatly diminished. On the 16th of August, 1876, the proportion of the red to the white blood-corpuscles was 3 to 2. On the 12th of October, 1876, after 414 pills (piperine, 5.0; ol. eucalypti, 10.0; quinine muriat., 2.0; ad pil. No. 100) had been taken, and the skin over the splenic tumor had been daily faradized, an accurate count gave the proportion 9 to 1. Afterwards the patient continued taking the pills outside the hospital for two months. On the 30th of March, 1878, the patient again presented himself to *Mosler*, who was astonished at his excellent condition and good appearance, saying he was going to purchase an inn and marry again. The sternum was painful in one spot only between the insertions of the fourth and fifth ribs; there was no longer any swelling, the splenic tumor had materially diminished. In one centimetre of blood there were 3,008,000 red, and 223,720 white blood-corpuscles (ratio 13 to 1). The patient now took:

℞ Tr. eucalypti,
Syr. chloroformati .... āā 25.0
Ol. eucalypti e foliis .... gtt. 25.0
M. One teaspoonful three times a day.

Cutler and *Bradford* (*American Journal of the Medical Sciences*, January, 1878, p. 84) saw the white corpuscles diminish under the prolonged use of arsenic, so that the proportion was not far from normal in a case of splenic leucæmia.

In the majority of women suffering from leucæmia, *Mosler* observed amenorrhœa. Profuse menorrhagia combined with intestinal hemorrhage occurred in two of his patients. In men also, as a rule, when the leucæmia has existed a long time, the genital functions are markedly disturbed. Exceptionally patients are harassed by involuntary seminal emissions, or by erection without a sensual feeling. *Peabody* has collected the histories of nine cases of priapism occurring in leucæmic patients, and adds one of his own. In his case, complete erection lasted for six weeks, while semi-erection existed for rather more than two weeks. It was confined to the corpora cavernosa, and the patient was able to micturate without difficulty. In most of the cases pain was a prominent symptom; priapism lasted from a few days to two months, or even longer. It has been attributed to thrombus of the small vessels of the penis, hemorrhage into the penis, and to nervous irritation.

SEASES OF THE BLADDER.—DISEASES OF THE URETHRA.—DISEASES OF THE PROSTATE. FUNCTIONAL DISEASES OF THE MALE GENERATIVE APPARATUS.

BY

ROBERT F. WEIR, M.D.,

OF NEW YORK,

TURER ON GENITO-URINARY DISEASES AT THE COLLEGE OF PHYSICIANS AND SURGEONS; ATTENDING
SURGEON TO THE NEW YORK AND ROOSEVELT HOSPITALS, ETC., NEW YORK.

DISEASES OF THE BLADDER.

GENERAL WORKS ON DISEASES OF THE URINARY ORGANS.

PICARD, H.: Traité des Maladies de l'urèthre. Paris, 1878.—PICARD, H.: Traité es maladies de la vessie. Paris, 1879.—CHACON, F. DE P.: Sur la région péri- éale. Paris, 1878.—PIRONDI, S.: Précis théorique et pratique des maladies des voies rinaires. Paris, 1878.—OTIS, F. N.: Stricture of the Male Urethra. N. Y., 1878.— ARRISON, REGINALD: Disorders of the Urinary Organs. London, 1878.—DELE- SSE: Pratique de la chirurgie des voies urinaires. Paris, 1878.—PAQUET, A.: ır la chirurgie clinique des maladies des voies urinaires. Paris, 1878.—VULPIAN, .: Maladies de l'appareil génito-urinaire et de ses annexes. Paris, 1879.—BOKAI, HANN: Krankheiten der männlichen Sexualorgane; Krankheiten der Blase. übingen, 1879.—THOMPSON, SIR HENRY: Clinical Lectures on Diseases of the rinary Organs (5th Ed.). London, 1879.—BUMSTEAD and TAYLOR: The Pathol- gy and Treatment of Venereal Diseases. New York, 1879.—KEYES, E. L.: Vene- eal Diseases, Including Stricture. New York, 1879.

I. MALFORMATIONS.

BIBLIOGRAPHY.

ENGLISCH, E.: Ueber angeborene Klappen und Divertikel der Harnröhre. Wien. ed. Presse, pp. 1373-1602, 1878.—OLIVER, T.: Absence of the Bladder. Lancet, ol. II., p. 829, 1879.

In a woman aged fifty-three, who had had for some time prior to her ath symptoms of cystitis, *Oliver* found a total absence of the bladder. he ureters, only one of which was pervious, opened directly into the ethra, which they joined at about one and a half inches from the meatus. ıe urethra itself only extended backwards beyond this a very short stance and terminated in a cul-de-sac. The pervious ureter was con- lerably dilated. The kidneys were the seat of lymphadenoma. *Oliver* otes a number of cases of absence of the bladder given in *Todd's* clopædia of Anatomy and Physiology; also one narrated by *Phillips,* ıere, in a female, the ureters opened through the abdominal parietes on ch side of the pubic region.

II. CYSTITIS.

BIBLIOGRAPHY.

1. Schüller, Max: Ueber die Localbehandlung des chronischen Blasen-Catarrhes. Berlin, 1877.—2. Cauvet, J.: Contribution à l'étude des accidents consécutifs à la déplétion brusque de la vessie. Paris, 1877.—3. Jurié, G.: Ueber den Mechanismus der Harnverhaltung bei Greisen. Archiv f. klin. Chir., XXI., p. 724, 1877.—4. Laforest, C. M.: Contributions à l'étude des cystites du col de la vessie. Paris, Thesis No. 289, 1878.—5. Thompson, Sir Henry: Cysto-phosphatic Concretions Formed in the Bladder. Lancet, Jan'y 12th, 1878.—6. Nunn, S. W., and Thornton, J. K.: Local Use of Solutions of Quinine in Chronic Irritation of the Bladder. Lancet, Feb'y 23d, 1878; June 1st, 1878.—7. Reliquet: Leçons sur les hemorhagies des voies urinaires. Gaz. des hôp., Nos. 48, 51, 52, 1878.—8. Teevan, W.: Chronic Cystitis after Lithotrity Cured by an Exclusive Milk Diet. Lancet, Vol. II., p. 80, 1878.—9. Miles, C. W.: Nitrate of Silver in Chronic Cystitis. American Practitioner, July, 1878.—10. Gross, S. W.: Symptoms and Treatment of Chronic Cystitis in the Male. Boston Med. and Surg. Journ., Nov. 14th, 1878.—11. Zucarelli, J. F.: Cystite traité par le chlorate de potasse. Thèse, Paris, No. 36, 1879.—12. Picard, H.: Des dangers du cathéterisme chez les vieillards. France méd., XXVI., Feb. 15th, 1879, et seq.—13. Dassein, H.: Extraite de stigmates de maïs dans les maladies de la vessie. Gaz. des hôp., Vol. 52, p. 100, 1879.—14. Deecke, Theod: On the Treatment of Chronic Catarrh of the Bladder and of Some Forms of Acute Cystitis. Buffalo Med. and Surg. Journ, Feb., 1879.—15. Simmons, D. B.: Quinine Internally in Cystitis. American Journ. Med. Sciences, April, 1879.—16. Jean, Alfred: Rétention incomplète d'urine, dans le cas de lésions prostatiques et de retrécissements de l'urèthre. Paris, Thesis No. 14, 1879.—17. Déspres, A.: Sur les conséquences du coït chez les vieillards et chez les urinaires. Rev. de thérap. méd. chir., p. 310, 1879.—18. Howe, J. W.: Injections of Linseed Oil for Chronic Cystitis. N. Y. Med. Journ., Vol. XXIX., p. 386, 1879.—19. Cazeneuve and Livom: On the Physiology of Vesical Epithelium. Journ. Anat. and Physiol., Jan., 1879.—20. Bramlett, W. H.: Cool Acidulated Water in Cystitis. Med. Record, Vol. XV., p. 141, 1879.—21. Weir, R. F.: On the Use of Catheters in the Treatment of the Hypertrophied Prostate. Hosp. Gaz., Vol. VI., p. 481, 1879.—22. Walsham: Suppurative Phlebitis Following Cystitis. Lancet, Vol. I., p. 665, 1879.—23. Thompson, Sir Henry: Remarks on the Production of Cystitis by Contagion through the Use of Instruments. British Medical Journal, Vol. I., p. 694, 1879.—24. Dufau: Stigmates de maïs dans les maladies de la vessie. Union méd., July 29th, p. 162, 1879.—25. Thompson, Sir Henry: Catarrh of the Bladder, Albumen in the Urine. Lancet, Vol. I., p. 6, 1880.—26. Gouley, J. W. S.: Cystorrhagia from Retention of Urine. Medical Record, XVII., p. 193, 1880.—27. Vauthier, J. Z. F.: Etude sur l'acide maizènique. 8vo, pp. 22, Brussels, 1880.

Schüller (1) draws a line between two classes of remedies for cystitis: those which arrest decomposition in the urine and those which tend to restore the mucous membrane to a natural condition. Carbolic acid does the former in one-half to two per cent solutions, but not the latter; salicylic acid, in one-third to one-half per cent solutions, acts well in both ways. Permanganate of potassa is also recommended in one-twentieth to one-fifth per cent, but it is apt to give rise to pain. Chloride of sodium in five per cent solutions is the most serviceable in muco-purulent catarrh. Nitrate of silver, in one-half to one per cent, and chloride of zinc, in one to two per cent solutions, are recommended in ulcerations of the mucous membrane and in cases attended with copious deposits of pus.

Jurié (3) contests the usually received opinion concerning prostatic retention of urine, and says that the hypertrophied prostate does not commonly grow into the cavity of the bladder. In several hundred cases of hypertrophied prostates which he examined, he saw such an increase in only eight or ten instances. The retention of urine is, therefore, not due to this enlargement of the prostate, but to the loss of power in the longitudinal muscular fibres of the bladder and prostate which, in a normal condition, open the mouth of the bladder. By the lengthening of the prostatic urethra, its walls are also more tightly applied to each other and thus offer an obstacle to the egress of urine. The retention of urine, he therefore thinks, is not to be ascribed to atony of the bladder nor to the valvular action of the prostate, but to the changes in the prostatic canal itself.

Thompson (5) says that the operation of lithotrity is occasionally followed by chronic cystitis with painful symptoms and by frequently recurring production of soft, mortar-like deposits, called by him "cysto-phosphatic." He considers that it is due to some destruction of the mucous membrane either by a traumatism of the operation or of the stone itself. Therefore, after such a bladder has been relieved of these incipient and frequently-recurring stones by either the flat lithotrite or by an aspirating catheter, it should be daily emptied and washed out with a solution of carbolic acid, one gr. to the ounce of water, or Condy's solution of permanganate of potash, six or eight minims to the ounce. It is especially to be observed, in connection with carbolized solutions, that they are not incompatible with any metallic salts that it may be desirable to inject immediately afterwards. After a thorough cleansing, the bladder walls are to be bathed with some astringent, such as the salts of silver, copper, or lead, beginning with weak solutions, which are gradually to be increased in strength. The nitrate of silver at first should not exceed one grain to four ounces of water, and sulphate of copper is used in the same strength. The medicated washing out can be repeated every other day.

Thornton (6), following the suggestion of *Nunn*—of injecting quinine into the bladder in chronic cystitis—uses a solution of two grains to the ounce of water, acidulated sufficiently with sulphuric acid to dissolve the salt. Three ounces are injected into the bladder, and, after a few seconds, two ounces are withdrawn, leaving the remainder in the bladder. Relief promptly followed in the two cases described.

Miles (9) has, from the report made by Dr. *J. M. Thompson* in the *Louisville Medical News* for June 1st, 1878, used injections into the bladder, when chronically inflamed, of solutions of nitrate of silver, but not in such strength as has been recommended by *Richardson*, of New Orleans (*i. e.*, twenty to sixty grains to the ounce of water). *Miles* uses two or three grains to the ounce, with an increase of two and a half grs. to the ounce at each injection. He rarely went beyond a ten-grain solution. If the pain subsequently experienced is great, it is ameliorated by an injection of sweet oil.

Dufau (13) uses the stigmates de maïs in extract and decoction, but prefers the former in a syrup. Its employment, however, should be avoided in acute cystitis. The best results are obtained in muco-purulent catarrh of the bladder with calculous formations. The secretion of urine is generally increased.

Vauthier (26) attests, as the result of experience, that the Zea maïs (spurred Indian corn) is of decided service in acute and chronic cystitis, but particularly in the catarrhal forms, and that it can be administered in the form of the maizenic acid, which he has discovered, or in the form of an infusion, one part to ten of boiling water, of which one tablespoonful is taken every two hours.

Deecke (14) recommends in cystitis the use of lactic acid injections of one-per-cent strength. It not only arrests decomposition of the urine, but also dissolves with rapidity the muco-pus and shreddy deposits in the urine. It moreover keeps in solution the earthy salts. It is advisable also to administer it internally conjoined with the free consumption of buttermilk. The doses given should range from one to two grammes per diem. He gives a record of twenty-one cases treated in this manner; usually it was only necessary to have recourse to two or three injections of the acid; in only one case, however, it was used ten times. He points out that this is probably one of the reasons of the efficacy of George Johnson's milk diet (see *Lancet*, 1876, Vol. ii., 847 pp.).

Howe (18) successfully treated a case of chronic cystitis by distending the bladder with linseed oil, which was retained as long as possible. This was done daily, eight ounces being injected at each sitting. The subsequent progress of the case is reported as satisfactory.

Déspres (17) speaks of the danger of coitus in elderly men affected with urinary diseases, and mentions among the symptoms an amygdalitis which he has also frequently seen in young married men and which he considers to be due to reflex action.

P. Cazeneuve and *Livom* (19) publish papers on the subject of the power of absorption of the mucous membrane of the normal bladder, of which the following is an abstract:

Various opinions are held as to the power of absorption possessed by the mucous membrane of the bladder. Some consider its absorptive power very great (*Segalas*); others think it very feeble (*Bérard, Demarquay*); and a third class deny its existence (*Kuss, Morel, Sussini*). Previous investigations have usually consisted in injecting poisonous drugs into the bladder and observing the results; but the authors of the present communication have adopted a new method of investigation, the principle of which is to establish whether or not any urea, the chief urinary ingredient, passes through the walls of the bladder. Their "modus operandi" is to tie the prepuce of a dog for some hours before the operation, so as to keep the urine in the bladder. They then expose this viscus, remove it full of urine by means of a ligature, and, after washing it externally with distilled water, they plunge it into three quarts of distilled water at a temperature of 25° C. From time to time the water outside is tested by means of hypobromite of soda, which indicates, by its effervesence, the presence of urea. In a series of twenty experiments it was found that it took from three to four hours for the urea to pass through, in the case of a bladder freshly removed; but in one taken out the previous evening, dialysis occurred in from ten to fifteen minutes. The results of their numerous experiments may be summed up as follows:

1. Desquamation of the vesical epithelium, brought about by any mechanical means, as from the blunt point of a sound, is followed by vesical permeability, and in this point they corroborate *Kuss*, who holds that the impermeability of the bladder is due to a peculiar property of the vesical epithelium.

2. The increase or diminution of the temperature of the body affects the characteristics of the epithelium, for, in an animal well fed, the resisting function of the epithelium is very marked, while in one that has been starved it lasts only a very short time after death.

3. Injuring the kidneys, or cutting the spinal cord, affected the physiological properties of the vesical epithelium in a very marked degree.

On the important subject of the proper treatment of a long-standing hypertrophy of the prostate several interesting papers have lately been presented, a summary of which is here given.

Picard (12) says that the introduction of a catheter should not be hastily determined upon, for it may produe, 1st, syncope; 2d, vesical contractions with coincident hemorrhage from the altered pressure on the bladder-walls; and 3d, nephritis. The tenesmus comes, he states, from the instrumental irritation of the vesical meatus as a centre, for it is wanting in the operation of aspiration. The hæmaturia is due, not only to the alteration in the pressure on the blood-vessels, but also to the compression of the folded or wrinkled mucous membrane by the muscular efforts of the bladder. He gives quinine before using the catheter, and does not entirely empty the bladder and immediately reinjects a quantity of antiseptic fluid sufficient to prevent the hemorrhage. While it may be too strong to say that advanced age is a contra-indication to the use of a catheter, yet he thinks it is a condition that demands all possible precautions.

It is for the purpose of avoiding further contusion of the mucous membrane of the bladder that *Reliquet* (7) recommends that ergot should not be administered in the retention of urine of prostatic origin.

Thompson (23) refers to the dangers of unclean catheters and cites instances wherein contagion was transmitted in this way from one patient to another. He gives the following recommendations:

Firstly: All metal instruments—catheters, sounds, and lithotrites—

after use, especially in cases of muco-purulent urine, should be plunged for a minute or two into boiling water, to which either a little common soda or a little carbolic acid has been added. If the boiling point of water be not considered absolutely sufficient, a strong solution of chloride of zinc in water may be used. At the strength of twelve per cent solution the boiling point is 220° Fahr., or eight above that of boiling water. He has for some years past, since the suggestion of *Rolleston*, placed all gum and other catheters and bougies in a bath of weak carbolic acid immediately after use.

Secondly: He has more recently—that is, since the occurrence described—added a solution of carbolic acid to the oil used for the lubrication of instruments. Oil being the remedial agent for the caustic effects of carbolic acid, there is no danger in applying to the urethra a comparatively strong solution of the acid in oil, since no irritating effect whatever is produced and the disinfectant influence is unimpaired.

He gives the following formula, which he guarantees is absolutely unirritating: ℞ Acidi carbolici med., gr. xxij.; olei olivæ, ℥ i.

A free use of this as a lubricant to all instruments before using, he believes, will insure, at all events, in combination with the modes of cleaning just described, safety from the occurrence of any contagion by means of instrumental treatment.

Gouley (26) thinks, with *Stapfer* and others, that the most common factor in the production of hemorrhage of the bladder, when the organ has been subjected to distention by reason of retention of urine, is the sudden taking away of the hydraulic support from the distended blood-vessels, and hence advises that the old direction of emptying the bladder only very slowly be more strictly adhered to, especially in cases of hypertrophy of the prostate—with the additional precaution that, as soon as half a pint of urine is evacuated, an equal amount of a strong solution of warm borax solution be immediately thrown into the bladder, then a pint of urine again drawn, and half a pint of borax water injected, and so on, for six or eight times, until the bladder contains a clear borax solution. This injection, as well as other washings, is best accomplished by means of a soft rubber reservoir and two-way stopcock attached to the catheter. When cystorrhagia prevails to a marked degree, a similar procedure should be resorted to, *i. e.*, an injection into the bladder of a small amount of borax solution and then the sucking out through the catheter, introduced to break up the clots, of only two or three ounces of blood by means of a Bigelow's rubber evacuator or by a syringe. The surgeon should continue at the same sitting to make alternate injections and aspirations, never withdrawing more than he has injected, so that the contents of the bladder will remain about the same, until all the blood has been removed and the returning liquid is nearly clear.

III. DIPHTHERIA AND TUBERCULOSIS OF THE BLADDER.

BIBLIOGRAPHY.

28. Theile, Max: Ueber Diphtherie der Harnorgane. Virchow und Hirsch's Jahresbericht, p. 233, 1877.—29. Girard, E. V. A.: De la Cystite pseudomembraneuse. Paris, Thesis No. 180, 1877.—30. Virchow, R.: Diphtherie und Tuberculose der Harnorgane. Charité-Ann., Berlin, IV., 774, 1879.—31. Picard, H.: Tuberculisation génitale et urinaire. Gaz. hebdom., XVI., 441, 472, 1879.—32. Bierry, L.: De la tuberculose primitive des voies urinaires. Paris, Thesis No. 359, 1878.—33. Guébhard, A.: Sur la cystite tuberculeuse. Paris, 8vo, 1878.—34. Tapret, O.: Etude clinique sur la tuberculose urinaire. Arch. gén. de méd., CXLIV., May and July, 1878. London Med. Record, May 15th, 1879.—35. Angagneur: Du tuberculose des organes génito-urinaires. Lyon méd., 4, 1878.—36. Terrillon: Tuberculose génito-urinaire. Prog. méd., VII., 446, 1879.—37.

The interest that has lately been developed concerning these varieties of cystitis has led to their separation from the preceding report.

Virchow (30), who presents seven cases of diphtheria affecting the urinary organs, says it may be confined to the bladder and that an alkaline condition of the urine is apt to promote its formation. Ulcerations may subsequently form and give rise to hemorrhage, and while similar destruction is met with in the pelvis of the kidney, yet it is rare to find the ureters involved, while, in the kidney itself, the glomeruli or tubules get choked up with the diphtheritic masses.

Theile (28) has collected ninety-one cases of urinary diphtheria, most of which originated from contagion from without, *i. e.*, by the catheter. In twenty-one cases, the infection could be traced from a wounded urethra or vagina; in sixty-four, it was supposed to have been introduced by the catheter. Indeed, nearly all the cases presented some condition that demanded the frequent use of the catheter, yet some occurred where this explanation would not apply, as for instance in five cases complicating typhoid fever or peritonitis, where an instrument was not used. Of nineteen cases where diphtheria involved the kidneys, in thirteen the bladder was also involved, in six the viscus escaped. No explanation is offered of this fact. The non-implication of the ureters is likened to the action of the vas deferens in epididymitis.

Girard (29) alludes to seven cases of this affection occurring under *Guyon*, only two of which recovered.

The contributions of *Guebhardt* (33) are based on the study of thirty-three cases of tuberculous cystitis which, for the greater part, were made at the hospital Necker under Professor *Guyon*. The author holds that there exist two principal classes of tuberculous cystitis: a primary one, which is not preceded by tubercles in any other organ; and a secondary one, which follows immediately upon tuberculosis of the lungs or the genital organs, and hastens the end. The first class can often remain confined for a certain time to the bladder without spreading to the rest of the organism, and then attack either the genito-urinary tract, or any other organ. The author insists especially on the difference between what is termed urinary tuberculosis, and genito-urinary tuberculosis. The latter is often met with, while the former is more rare and localized in the bladder, urethra, and the kidneys, without spreading to the genital organs. At the *post-mortem* examinations, granulations, which are first gray, then yellow, are always found in the bladder, as well as characteristic ulcerations, either isolated or in groups, of various sizes. These lesions always begin at the neck of the bladder, and spread thence to the urethra, the prostate gland, the ureters, and the kidneys. The pain is sometimes almost excruciating, and often takes the form of neuralgia; the other symptoms are urethral and vesical spasms and hæmaturia. Although these phenomena are not pathognomonic, still they may often help towards making a diagnosis in most of the cases. The treatment consists in painting the neck of the bladder with a weak solution of nitrate of silver.

The most complete description of this form of urinary phthisis, which is particularly interesting in its group of vesical symptoms is by *Tapret* (34), who considers urinary phthisis as belonging to the class of affections which Pidoux designated under the name of anomalous phthisis. He thinks that the symptoms by which it shows itself are sufficiently characteristic to enable the practitioner to make a correct diagnosis, and consequently to adopt a rational treatment. So far as regards the etiology of the disease, urinary tuberculosis is essentially an affection of adult age; it seldom occurs in females, but appears in males between the age of twenty and forty-five. The symptoms present some interesting variations, according as the disease is principally confined to the kidneys, the bladder, the prostate gland, or the urethra.

PRIMARY OR ISOLATED TUBERCULOSIS OF THE KIDNEY.—The symptoms of this affection (hæmaturia, albuminuria, pus in the urine, combined or not with polyuria, spontaneous pains, or pains caused only by pressure on the lumbar region) are not characteristic, and might as well be attributed to other renal affections, such as interstitial nephritis, gravel, etc., thus rendering the diagnosis very difficult. The progress of the affection is generally slow, except in cases where the tubercles develop more rapidly, when death occurs generally at the end of a few months. The patient dies of uræmia, owing to the destruction of the renal tissue.

PRIMARY OR ISOLATED TUBERCULOSIS OF THE BLADDER.—*Tapret* thinks that there really exists a tuberculous cystitis, which has hitherto escaped observation, because attention had been principally directed to the kidneys, and the general opinion has been that lesions of the bladder occurred only in very exceptional cases or in the last stages of the disease.

The characteristic symptoms of this cystitis are: hæmaturia, appearing at an early stage, so-called premonitory hæmaturia, polyuria, which only shows itself at irregular intervals, and owing to divers causes; pains in the region of the neck of the bladder and a peculiar tenderness of the bladder, the latter being almost always irritable, and in a permanent state of contraction. In the few cases where it has retained its normal capacity, it is sometimes possible, by passing a sound into the bladder, or rectal examination, to feel a hardened spot on the fundus of the organ. This tuberculous cystitis generally progresses very slowly, and ends in consumption or urinary phthisis unless some local accident should bring on a cachectic state more rapidly.

PRIMARY OR ISOLATED TUBERCULOSIS OF THE PROSTATE GLAND.—This embraces two distinct clinical forms of the affection, a rectal or circumferential form and an urethral or urethro-cystic form. The latter presents the symptoms which are generally attributed to cystitis or tuberculous urethritis, pains during micturition, and while the catheter is being used, blennorhagia, prostatorrhœa, spasmodic retention of urine. During the latter stages of this affection, fistulas generally form, opening from the prostate gland into the urethra, the rectum, or the perineum. According to *Tapret*, all these various symptoms of the presence of tubercles in the genito-urinary organs only acquire importance after the urinary tracts, and especially the neck of the bladder, have been invaded by tubercles. When the neck of the bladder has been reached, the disease assumes a characteristic appearance, which is typical, and may be thus briefly defined. An individual, aged from twenty to forty, who has hitherto enjoyed good health, suddenly sees hæmaturias appear without any special cause, and without pain; these are followed after an interval of time, varying according to the individual, by retention of urine, which, however, is easily overcome; then the desire to micturate begins to grow more and more frequent and imperious. The act itself is very painful; the patient passes, with a great deal of pain and trouble, a few drops of urine leaving a deposit of blood-streaked pus in the vessel. At intervals the urine is more abundant, clear, almost normal (nervous urine), or dusky and discolored (in deeply-seated diseases of the kidney). There is also blennorrhœa of the deep parts of the urethra; the bladder is either small or dilated, the neck painful, the fundus hardened, no evidence of foreign bodies; the renal region is tender to the touch and the prostate gland presents a knotted appearance. The general state of health of the patient remains for a long time satisfactory. His temperature is hardly ever raised or his digestion impaired. The principal complaint is that he cannot stand upright without suffering. The progress of the affection is, though slow, yet sure to be fatal, and death is due either to the urinary phthisis, or to a complication of acute pulmonary phthisis.

IV. CYSTOTOMY, ETC., FOR CYSTITIS.

BIBLIOGRAPHY.

37. Bottini, E.: Radical-Behandlung der auf Hypertrophie der Prostata beruhenden Ischurie. Arch. f. klin. Chir., XXI., 1, 1877; Practitioner, Nov., 1877.—38. Teevan, W.: Cystotomy for Cystitis. Lancet, I., p. 478, 1879.—39. Thompson, Sir Henry: An Operation for the Relief of Long-Standing Hypertrophy of the Prostate. Med. Times and Gaz., I., p. 710, 1879.—40. Barss, I. R.: Cystotomy for Cystitis. Brit. Med. and Surg. Journ., CI., p. 797, 1879.—41. Howe, I. W.: Chronic Cystitis treated by perineal Section and Dilatation of the Neck of the Bladder. N. Y. Med. Journ., XXIV., p. 385, 1879.—42. Teevan, W. F.: Lettsomian Lectures on the Treatment of Stricture of the Urethra, Enlarged Prostate, and Stone in the Bladder. Lancet, April 17th, 1880.

Bottini (37) proposes to treat the difficulty commonly experienced in passing water in cases of hypertrophy of the prostate gland by the galvano-caustic method. He recommends, according to the kind of the hypertrophy, either the galvano-caustic erosion or simple section of the enlarged lobule. The galvano-caustic destined for erosion, is like the well-known prostatic catheter of Mercier. The thermo-galvanic incisor resembles a lithotrite, the male arm of which is composed of a platinum blade, which is attached to the staff by means of copper points, and glides in the glass appendix of the female arm. The points of the instrument must, when in use, be firmly pressed against the lobe requiring division. Neither the process of erosion nor of division occasions much pain, so that anæsthetics are not required. The urine soon begins to flow after the operation, however severe may have been the previous strangury. No bad results have hitherto followed the use of the instruments, and not even vesical catarrh has been observed, though the urine continues to be bloody for a little while. Galvano-caustic erosion is best adapted for partial and only slightly projecting swelling of the supra-collicular part of the gland. On the other hand, galvano-caustic fusion is more appropriate for cases where the whole gland is enlarged, or where particular lobes have undergone great hypertrophy. In such cases it is advisable to make the instrument press against the depression between two adjoining elevations, rather than against the projecting mass. The contra-indications against this mode of operating are inactivity of the detrusors, highly abnormal condition of the urine, and coincident extensive disease of the kidneys.

For an obstinate cystitis following lithotrity, *Teevan* (38) did cystotomy by a median incision in the perineum, incising, however, the neck of the bladder vertically with a probe-pointed knife to the depth of about half an inch. The wound closed in six weeks. The relief was immediate and permanent.

Bryant stated, in the discussion that followed, that he had performed cystotomy for cystitis six times, in three of which relief followed, and in the other three death occurred from prostatic or renal disease.

Thompson (39) recommended in those cases where a patient was unable to pass water by his own efforts and had to use the catheter with great pain and difficulty, the insertion of a tube *en permanence* in the bladder, by making an opening just above the penis. This might be done safely by means of a staff, as in the high operation for stone. But as in these cases the bladder was small and contracted, it was to be opened rather behind than above the pubis, so as to make certain not to injure the peritoneum. He had resorted to this in three cases. *Weir*, in 1876, called attention to this operation which was originally done by *Van Buren*, of New York, who put it into effect some ten years previously.

Barss (40) performed, for a long-standing cystitis, lateral lithotomy, as suggested by *W. Parker*, of New York, and *Bickersteth*, of Liverpool, in 1867. The opening was kept patent by a tube introduced from the perineum into the bladder, and to this was attached a receptacle for the urine. By the operation he was very much relieved, though not cured; eighteen months later he died from renal trouble, the bladder having been kept drained till then.

Howe (41), in a case of severe cystitis of six months' duration in a man of eighteen, submitted his patient to the median section for stone, and dilated the prostate and vesical orifice sufficiently to admit the index and middle fingers. The patient made a rapid recovery. Dr. H. reports two other cases similarly operated on by him with equally satisfactory results.

Teevan (42) advises, for the severer forms of cystitis from hypertrophy of the prostate, a suprapubic puncture, or, what he prefers, an opening in the perineum at the apex of the prostate, through which a catheter can be readily introduced into the bladder.

V. TUMORS OF THE BLADDER.

BIBLIOGRAPHY.

43. COUPLAND, SIDNEY: (a) Lymphosarcoma of the Prostate; (b) Medullary Cancer of the Prostate. Trans. Path. Soc. of London, Vol. XXVIII., pp. 179–185, 1877.—44. KRABBEL: Blasenkrebs. Archiv f. klin. Chirurg., XXIII., p. 520.—45. STIMSON, L. A.: Sarcoma of the Bladder. Medical Record, Nov. 16th, 1878.—46. PLANTY-MAUXION, EMIL: Des kystes de la prostate. Paris, Thesis No. 127, 1878.—47. NORTON, A. T.: Excision of Papilloma of the Bladder. Lancet, May

24th, 1879.—48. Mynter, H.: New Growth in the Bladder of a Child. Buffalo Med. and Surg. Journal, Vol. XIX., p. 9, 1879.—49. Le Dentu: Un kyste de la prostate. Bull. et Mem. Soc. Chirurg. de Paris, V. I., p. 27, 1879.—50. Nitze, Max: Eine neue Beobachtungs- und Untersuchungsmethode für Harnröhre, Harnblase, und Rectum. Wien. med. Wochenschr., XXIX., p. 649.—51. Thompson, Sir Henry: The Nietze-Leitner (sic) Mode of Examining the Bladder. Lancet, I., p. 713, 1879.—52. Humphrey, G. M.: Tumor of the Bladder Removed by Perineal Incision. Trans. Medico-Chir. Soc., 1879, p. 422.—53. Heath, C.: Villous Sarcoma of the Bladder. Death. Med. Times and Gaz., II., p. 662, 1879.—54. Thomson, J. R. M.: Hydatid of Bladder. Australian Med, Journal, I., p. 425, 1879.—55. Hudson, R. S.: Villous Disease of the Bladder and its Surgical Treatment. Dublin Journ. Med. Sciences, p. 490, 1879.—56. Langhans, T.: Cavernöser Tumor der Blase. Arch. f. path. Anatom., Berlin, p. 291, 1879.

Coupland (43) found at the autopsy of a male, æt. 19, whose bladder symptoms during life were severe, and in whom a rectal examination showed the prostate to be so large as almost to protrude through the anus, that the growth was about the size of the two fists, and involved to an equal extent both sides of the prostate. Some soft white nodules projected into the bladder. The lumbar and pelvic glands were also enlarged. Microscopically, it was shown to be medullary cancer. His second case was encountered in a man æt. 29, who had symptoms of stone. No rectal enlargement of the prostate was detected. At the autopsy the base of the bladder was occupied by a large fleshy mass continuous with the prostate, and projecting into the bladder just below the ureteral orifices. The whole prostate was the seat of a new growth. There was no involvement of the pelvic, lumbar, or inguinal lymphatic glands, but secondary deposits were found in the pancreas and suprarenal capsules. Microscopically, the growth was that known as lymphosarcoma.

In *Stimson's* (45) case, a diagnosis was not made, as the patient was almost *in extremis* when first seen. At the autopsy, a sarcomatous tumor, nearly three inches in diameter, was found attached to the posterior wall of the bladder, about four inches from its outlet. It had a pedicle not quite as thick as one's forefinger. The tumor was one that could easily have been diagnosticated and removed, had the patient been seen earlier.

Planty-Mauxion (46), besides his own case, has only been able to collect three others of hydatid of the prostate: one of these was from *Curling*, with an uncertain history; another from *Lowell*, with a fatal termination, and a third from *Mallez*, wherein a correct diagnosis was made, and recovery took place. His own case was as follows: A man of twenty years, having a history of urinary difficulties lasting over a year, had a swelling in the left hypochondrium, the size of a seven months' fœtal head. By the rectal examination, a fluctuating tumor was found, occupying the region of the prostate. Punctures here gave exit respectively to 300, 800, and 60 cc., and a microscopical examination of the fluid showed the hooklets of the echinococci. The later punctures became purulent, and then a drainage tube was introduced into the tumor, which occupied the right lobe of the prostate, and its cavity was washed frequently with iodinized water. After a week, the left lobe was similarly treated. Two months later, the patient was nearly well, though the prostate was still enlarged. The author believes that cysts quite frequently occur in the prostate, but are not noticed during life on account of the smallness of their size, and that hydatids can only be recognized by their large size and distinct fluctuation. Puncture will always determine their character. Puncture and incision are not sufficient for cure, and ought to be combined with injections of tincture of iodine.

At a meeting of the Clinical Society of London, *Norton* (47) read the notes of a case of papilloma of the bladder. A female, thirty-four years of age, was admitted into St. Mary's Hospital, suffering from the effects of long-continued hemorrhage from the bladder. The urine contained also much mucus and phosphates, masses of the latter being frequently passed. There was great pain after micturition, and constant desire to pass water. No calculus could be found, but the bladder was thick in the region of the trigone, and a digital examination per urethram under chloroform confirmed the diagnosis of a tumor. The growth was one inch square, slightly raised, and coated with phosphatic deposit. Its removal was decided upon, the alternative to the patient lying between the risk of a severe operation, and the continued pain, and a possible early fatal hemorrhage or blood-poisoning. It was impossible to remove the growth through the urethra, and it was decided to cut away the mass by opening the vagina. The spring scissors were inserted, one blade into the bladder nearly up to the tumor, and the other into the vagina, and closed; the front wall of the vagina was then incised centrally to within half an inch of the uterus, and the vaginal wall was dissected from the bladder; the growth was then seized with the volsella forceps and drawn forwards, excised by the scissors and removed. Bleeding was arrested by the actual cautery, and the lateral flaps of the vagina approximated by sutures. To prevent further hemorrhage, a catheter was inserted, and the bladder compressed by plugging the vagina. No bleeding of importance took place. The temperature remained below normal, and the pulse rose to 120. Severe vomiting was persistent until the tenth day after the operation, notwithstanding subcutaneous injection of morphia and five-grain doses of quinine administered frequently by the stomach. After the tenth day she was considered out of danger, was making good progress, took food well, and was cheerful; but two days later, after vomiting, she fell asleep, and died in sleep from syncope. On the post-mortem examination the heart was found to be healthy, and its left side empty. The blood was mostly fluid. The wound was sloughing on the surface, with some phosphatic deposit around it and the orifices of the ureters. The vesical mucous membrane was congested, but of normal consistence. No peritonitis, and no thrombosis. The examination showed that, so far as the peritoneum was concerned, a tumor nearly twice the length and breadth could have been removed through the wound, but the ureters would have been included in such an operation. Whether or not such inclusion of the ureters would add to the severity of the operation cannot be proved, but it is probable that the urine would escape without injury to the parts around. A microscopical examination showed the tumor to be a papilloma. Since the above case, Mr. Norton had operated upon a second case of tumor of the bladder, with a successful result.

Mynter (48) gives an instance of fibrous polypi of the bladder, encountered in a female child sixteen months of age, who in vesical tenesmus extruded a fleshy mass from the meatus urinarius. The urethra was dilated sufficiently to admit the finger, and a mass, the size of a pigeon's egg, was removed, but many other smaller ones were felt attached generally to the bladder-walls. These were not removed. The subsequent history is not recorded. Microscopically the polypus was a fibroma.

Le Dentu (49) presented to the Society a specimen, obtained from a man who, during life, had hæmaturia and difficulty in urination, which showed, aside from the usual hypertrophic changes in the bladder, a projection into this organ in the site of the middle lobe of the prostate. It with the whole inferior part of the prostate was converted into a large cyst the size of a small orange, which extended from the apex to the vesiculæ seminales. The ejaculatory ducts were outside of this cavity, which was moreover unilocular. It was thought to be a cyst, resulting from obliteration of some of the ducts of the prostate gland. For the explanation offered by *Le Dentu* might be substituted that of *Englisch*, of obliteration of the duct of the utriculus, and its subsequent distension. *Englisch* believes this to be a not infrequent cause of retention of urine in the new-born child.

Nitze (50) has invented an apparatus by which an electric light can be carried to the bottom of an endoscopic tube, and by the illumination thus produced the observer is enabled to distinguish objects in the bladder quite readily. Overheating is prevented by a current of water kept passing outside the tube containing the incandescent platinum wire. By this contrivance, known as the Nitze-Leiter endoscope, *Thompson* (51) is led to believe that we may perhaps have a more positive means of diagnosticating villous tumor of the bladder, or an encysted calculus, or possibly in the detection of the nature of foreign bodies in the bladder. All other conditions can as well, and perhaps better, be determined by older instruments and methods of investigation.

Humphrey's (52) case is one of much interest. It was a fibro-sarcoma situated in the anterior wall of the bladder in a man of twenty-one years. A bimanual examination completed the diagnosis. By lateral cystotomy the mass was reached, and a tumor, the size of an orange, was removed. From causes unknown, the patient suffered greatly for two months, and then recovered. *Humphrey* advises, in cases of doubtful diagnosis, to make the ordinary perineal incision and a digital exploration of the bladder.

Thompson (54) describes a tumor which appeared on the left side of the hypogastric region, apparently attached to the bladder, in a man of forty-four, who had only frequent micturition with normal urine. The tumor was tense and non-fluctuating. Per rectum the prostate was normal, but above it could be felt a hard tumor. An attempt was made to thrust a trocar into the tumor, above the pubis, but the instrument, from a defect, failed to enter. As the result of the pressure, however, the tumor disappeared, nor by the rectal examination could it longer be detected. The patient urinated immediately thereafter, and the fluid thus obtained, on being examined, was found to contain the hooklets of the echinococcus. In a week his urination became natural, and he was discharged from the hospital.

Langhans (56) gives the post-mortem examination of a young man of nineteen, who had suffered with periodical hæmaturia for ten years, the last attack of which, having continued for ten days, resulted in his death. Cavernous angiomata were found involving several portions of the bladder-walls, the largest of which was found posteriorly just above the left ureter, and was $1\frac{1}{2}$ inches in diameter and $\frac{1}{4}$ inch thick.

VI. STONE IN THE BLADDER.

BIBLIOGRAPHY.

57. Bouloumié, P.: Spontaneous Fracture of a Calculus. Union méd., No. 18, 1877.—58. Henriet, Leon: Sur le traitement des affections calculeuses chez l'homme par la lithotritie. Paris, 8vo, 1877.—59. Lemaire, S.: Calcules enkystés de la vessie. Paris, Thesis No. 543, 1877.—60. McGregor, J. R.: Extraordinary Case of Urinary Calculi. Am. Journ. Med. Sciences, Jan., 1877.—61. Ord, W. M.: Clinical Lecture on the Examination of Urinary Calculi. Canada Journ. Med. Sciences, Vol. III., No. 9, 1878.—62. Thompson, Sir Henry: On the Use of the Microphone in the Bladder in the Search for Minute Concretions. British Medical Journal, June 8th, 1878.—63. Hill, Berkeley: The same. British Med. Journ., July 13th, 1878.—64. Ord, W. M.: Clinical Lecture on some Urinary Calculi and Urines. British Medical Journal, Sept. 7th, 1878.—65. Ord, W. M.: Spontaneous Disruption of Calculi within the Bladder. Trans. Path. Society, London, Vol. XXIX., p. 261.—66. Ord, W. M.: Indigo Calculus. Trans. Path. Soc., London, Vol. XXIX., p. 159.—67: Bigelow, H. J.: Litholapaxy. Am. Journ. Med. Sciences, Jan., 1878.—68. Weir, R. F.: Litholapaxy. Am. Journ. Med. Sciences, Jan., 1880.—69. Thompson, Sir Henry: Lithotrity at a single Sitting. Brit. Med. Journ., II., p. 161, 1879, and Lancet, I., p. 78, 1880.—70. Keyes, E. L.: Rapid Lithotrity. Am. Journ. Med. Sciences, April, 1880.—71. Bigelow, H. J.: An Improved Evacuator. Boston Med. and Surg. Journ., p. 30, 1880.—72. Guyon: Des modifications du jet d'urine. Gaz. des hôp., LII., p. 812, 1879.—73. Nepauer, J.: Beitrag zu der Lehre von der Harnsteinbildung in der Blase bei Kindern. Centralztg. f. Kinderheilk., Aug. 15th, 1879.—74. Gaujot: Calculus of Cystin (weighing 28 grammes). Bull. et Mem. Soc. de Chirurg., p. 177, 1878.—75. Southam, F. A.: Cystin Calculi (Four Cases). Brit. Med. Journ., II., p. 13, 1878.—76. Ultzmann, R.: Ein Beitrag zur Cystinurie. Wien. med. Presse, No. 29, 1878.—77. Harrison, R.: Large Cystin Calculus (weighing 1050 grs.). British Med. Journ., I., p. 71, 1880.

McGregor (60) had a patient, a woman aged sixty-three, who had passed for some four years several hundred calculi per urethram. By a vaginal examination the bladder was felt to be full of similar concretions, the mass pressing down on the superior wall of the vagina. The urethra was dilated, and an attempt made to extract the calculi, but it did not succeed. The finger was introduced, but apparently the bladder was empty, though the calculi could plainly be felt in what was supposed to be a sacculus, above the bladder. This was incised, as the opening in it could not be found, and several calculi removed, but it was then ascertained that a very large stone was present, whose removal was deemed unwise. Peritonitis ensued, and the autopsy showed that the calculous mass was contained in a displaced kidney, resting upon the bladder. On section of what remained of the kidney, there were removed five hundred and twenty calculi, from the size of a mustard seed to an almond, also a huge calculus, weighing fifty-one ounces, and measuring 6⅜ inches in length, and in the greatest circumference 16⅝ inches. Its composition was apparently (as no section was made) uric acid.

Ord (66) found, without any history, a beautiful specimen of indigo calculus, weighing forty grains. It was a flat concretion of a blue-black color, with a gray section. It left a blue mark on paper. It answered to the various tests of indigo, the most ready test of which, he states, is sublimation. This is the first calculus of its kind on record. A résumé is given by the observer on the subject of indigo formation in the economy. According to *Jaffe*, indigo is supposed to be derived from indol, which is formed when albumen is decomposed by a strong alkali, and in the body, when the peptones are broken up by the pancreatic juice. It is also found in the urine after a ligature has been applied to the small intestines in man when obstruction of the bowels exists—also in cholera. *Jaffe* has shown, in addition, that indigo is formed in an exclusively vegetable diet. *Olding* has also demonstrated that the dark color met with in the urine, from the use of creasote or carbolic acid, is due to the indigo present.

Bigelow (67). Although it is somewhat foreign to the purpose of this compendium to dwell at any length upon operative procedures, yet a space must be made for this, the most important advance in the last decade in the treatment of stone in the bladder. It is, in fact, the removal of stones from the bladder, not only in one sitting by crushing, but stones of a size beyond what had hitherto been removed by lithotrity. Notwithstanding this rapid removal had years ago been accomplished by *Heurteloup* and others, who demonstrated that the bladder would bear much rough usage, provided that all irritants in the way of fragments were removed, yet it was reserved to *Bigelow*, not only to show how it could best be done, but also, as essential to the success of the new operation, how the tedious pulverization and evacuation of the calculus could be avoided and the urethra saved from the mishaps of laceration in the withdrawal of debris-laden lithotrites. The latter was accomplished by the use of a large aspirating tube or catheter. Dr. *Bigelow* crushes in one sitting, under ether, by means of a strong lithotrite, stones reaching as high as two and one-quarter inches in diameter, and then sucks out the fragments through large (No. 28 to 32 Fr.) catheters, straight or curved, as is deemed best fitted to the individual case, by means of an india-rubber exhaust-bottle, at the bottom of which is a glass receptacle for the fragments to collect in and so escape regurgitation into the bladder.

Thompson (69) has adopted the method, and Drs. *Weir* (68) and *Keyes* (70) have presented summaries of the cases treated up to date. Dr. *Weir's* cases numbered 77, with a mortality of one death in 19 cases. and *Keyes*, three months later, was able to collect, including *Weir's*, 107 cases, with 6 deaths, or one death in about 18 cases. Several of the deaths may be ascribed to inexperience on the part of the operator rather than to the operation itself. So far, an unusual success has attended the operation. It is, however, one that should only be performed by a surgeon having some experience in instrumental manipulation of the bladder.

Guyon (72) calls attention to the value of the arrest of the stream of urine as a symptom of stone, provided only that the patient is in a standing position and that he is not an old person. It can only take place with a small stone, which then acts like a cork in a bottle.

Nepauer (73) considers that stone in the bladder is due to a settling of the precipitable salts conjoined with an obstacle preventing the precipitated mass being voided with the urine. He also, in his paper, explains the comparative infrequency of stone in the bladder in girls to the ready escape of a renal calculus through the female urethra, not because of its shortness or width, but by reason of the mouth of the bladder being, in girls, more positively the lowest part; this, together with the slighter depth of the bladder, forces, even in moderate contraction of the organ, the calculus into the urethra.

From the absence, in many cases, in the section of a mulberry calculus of any layers of triple phosphates, he is also led to conclude that the roughness and weight of a stone do not, *per se*, give rise to cystitis; that, in other words, the cause of the vesical inflammation present is not to be ascribed to the calculus itself.

VII. GONORRHŒA AND ITS SEQUELÆ.

BIBLIOGRAPHY.

78. Hadden, A: Congestive and Organic Stricture of the Urethra, Accompanied by Retention of Urine, Treated Successfully with Water-Pressure by Means of a Syringe. Chicago Med. Times, Aug., 1877.—79. Dowling, C.: Is Gleet Communicable? Clinic, Mch. 3d, 1877.—80. White, J. W.: Diagnosis of Urethral Strictures by Bulbous Bougies. Philadelphia Medical Times, May 26th, 1877.—81. Lefort, Léon: Note sur un manœuvre permettant souvent de franchir des rétrécissements dits infranchisable. Bullet. gén. de thérap., June 30th, 1877.—

82. Isenschmid, J.: Ueber die Recidive bei Harnröhren-Strictur. Correspondenzblatt der Schweizer Aerzte, II., 1877.—83. Duplay, S.: Cystite du col, spasme, contracture douloureuse de la urèthre. Progrès médical, No .52, 1877.—84. Hovell, F. M.: Oiling the Urethra. Medical Times and Gaz., Sept. 29th, 1877.—85. Golding, F. F.: Water-Pressure in Retention of Urine. New York Med. Jour., Nov. 10th, 1877.—86. Desnos, M.: Gonorrhœal Rheumatism of the Heart. Medical Times and Gaz., Nov. 24th, 1877.—87. Faucon: Peritonitis and Subperitoneal Phlegmon of Gonorrhœal Origin. Medical Times and Gaz., Nov. 24th, 1877.—88. Gosselin: Rheumatisme Blennorrhagique. Gaz. des hôp., 108, 1877.—89. Gould, A. P.: Why is Organic Stricture Most Common in the Bulbous Portion of the Urethra? Lancet, Dec. 8th, 1877.—90. Fauconnier, L.: Contribution à l'étude de l'urêthrite chronique simple chez l'homme, considerée surtout au point de vue anatomo-pathologique. Paris, Thesis, 1877.—91. Hardy: Gonorrhœal Rheumatism. Medical Times and Gaz., Feb. 2d, 1878.—92. Pirocchi, P.: On the Use of Tayuya in Phagedenic and Scrofulous Ulcers and Blennorrhagia. London Medical Record, Feb. 15th, 1878.—93. Gurjun Balsam in Gonorrhœa. London Med. Record, May 15th, 1878.—94. Borggs, C. S.: Stricture of the Urethra from Prolonged Masturbation. Nashville Med. and Surg. Jour., May, 1878.—95. Gross, S. W.: On the Rational Treatment of Stricture of the Urethra (with discussion). Medical Record, June 15th, 1878.—96. Déspres, A.: Epididymitis. Bull. de l'académie, No. 42, 1878.—97. Terrillon: Ruptures de l'urèthre. 8vo, Paris, 1878.—98. Clugnet, J. A.: Etude sur la vaginite aiguë. Paris, Thésis No. 270, 1878.—99. Gross, S. W.: Causes and Treatment of Gleet. Medical Record, Oct. 5th, 1878.—100. Gay, I.: Cathetherism in Cases of Stricture on Physiological Principles. Lancet, Nov. 16th, 1878.—101. Brinton, John H.: Pathology of Urethral Stricture (with Discussion). Philadelphia Medical Times, Dec. 6th, 1878.—102. Braune: Ueber Dislocation der Harnblase bei der Simon'schen Rectalpalpation. Verhandl. d. deutsch. Gesellsch. f. Chir., Berlin, VII., 1878.—103. Spire, E.: Du spasme de l'urèthre symptomatique. Paris, Thesis No. 475, 1878.—104. Hanot, V.: Orchitis in Typhoid Fever. London Medical Record, Dec. 15th, 1878.—105. Reliquet: Leçons sur les maladies des voies urinaires. 8vo, Paris, pp. 144, 1878.—106. Hill, Berkeley: Perineal Fistula Cured by Division of the Meatus, p. 92, 1879.—107. Sands, H. B.: Spasmodic Stricture. Hospital Gaz., VI., 1879.—108. Otis, F. N.: Urethrismus or Chronic Spasmodic Stricture. Hospital Gaz., VI., p. 99, 1870.—109. Esmarch, F.: Ueber Harnröhrenkrampf. Arch. f. klin. Chirur., XXIV., p. 589, 1879.—110. Wyeth, J. A.: Urethritis Treated by Cold. Hospital Gaz., V., 167, 1878.—111. Weir, R. F.: Urinary Infiltration and Abscess. Medical Record, XVI., p. 457, 1879.—112. Billroth, Th.: Harnröhren-Stricturen. Chirurg. Klinik, 1871–76, p. 322, Berlin.—113. Otis, F. N.: Contracted Meatus as a Cause of Gleet and Retention of Urine. Hospital Gaz., V., p. 106, 1879.—114. Zeissl: Zur Therapie des Trippers der männlichen Harnröhre. Wien. med. Wochs., Sept. 20th, 1879.—115. Godin, E.: Sur l'urèthrite d'origine rheumatismale. Paris, Thesis No. 322, 1879.—116. Grünfeld, I.: Ueber Erosionen und Geschwüre der Harnröhre. Wien. Med. Doct. Coll., V., p. 1, 1879.—117. Grégory, W.: De la méthode sanglante dans les retrécissements de l'urèthre. Paris, Thesis No. 295, 1879.—118. Duncan, M. J.: Clinical Lecture on Retention of Urine (in Women). Medical Times and Gaz, Oct. 18th, 1879.—119. Verneuil: De l'orchite à forme névralgique. Journ. de Conn. méd. chir. prat., 202, 1879.—120. Hill, Berkeley: Clinical Lecture on Stricture of the Urethra. Brit. Med. Jour., Nov. 15th, et seq., 1879.—121. Gay, C. C.: On the Employment of the Catheter; Suggestions for Overcoming some of the Difficulties of its Use. American Practitioner, XX., 193, 1879.—122. Sabourin, C.: De la retention passagère de l'urine après les traumatismes non-chirurgicaux. Archiv gén. de méd., CXLIV., Oct., 1879.—123. Mastin, C. H.: Stricture of the Prostatic Urethra. Boston Med. and Surg. Jour., C. 878, 1879.—124. Dugas, L. A.: Acute Orchitis Treated by the Roller-Bandage. New Orleans Med. and Surg. Journ., VII., 683, 1879.

—125. Terrillon and Schwartz: Contributions expérimentales à l'étude de la pathogénie de la vaginalite. Gaz. méd. de Paris, I., 385, 1879.—126. Guyon: Etiologie des retrécissements de l'urèthre. Journ. de méd. et chir. prat., LI., 12, 1880. 127. White, O. A.: Mechanical Compression in Orchitis. Boston Med. and Surgical Journ., 99, 1880.

White (80) criticises *Gross's* paper on stricture following masturbation and takes the view that this is very rarely a cause for stricture of the urethra, and that *Gross's* narrowings, which were single and generally at the same depth in the canal, were due to the bougie à boule catching in the anterior layer of the triangular ligament. *White* made several dissections with the bougies à boule caught in this locality and proved this point.

Desnos (86) presented a case of gonorrhœal rheumatism of the heart to the Paris Hospital Society, and observed that, although these cases were rare, and usually pass unperceived, yet their true nature and connection are sometimes brought to light by means of the intervening arthritis. About a month ago, the subject of his case came into the hospital with acute bronchitis, the heart being then in a normal condition. Some days after, the patient was seized with pain in the shoulder, which then localized itself in the sterno-clavicular articulation, and it was then discovered that he had gonorrhœa. All of a sudden he was seized with violent palpitation of the heart.

The diagnosis was made of narrowing and insufficiency of the mitral orifice, and œdema and infiltration of the inferior extremities, without albumen in the urine, supervened. At the autopsy, a small ulcer was found on the mitral valve, together with a considerable vegetant endocarditis of the aortic valves and the whole of the interior of the heart.

Faucon (87) arrives at the following conclusions: 1. Peritonitis and subperitoneal phlegmon should be ranked amongst the possible complications of gonorrhœa. 2. These accidents are only distinct effects of the blennorrhagic inflammation, propagated from the urethra to the peritoneum or the subperitoneal cellular tissue by the intermedium of the vas deferens, the vesiculæ seminales, the prostate (perhaps the bladder, ureters, and kidneys), and the cellular "atmosphere" surrounding these organs. 3. Their appearance is, therefore, always preceded by the ordinary blennorrhagic complications resulting from the preliminary inflammation of the tissues or organs which serve as intermedia (deferentitis, vesiculitis, etc.), so that they are true tertiary accidents of gonorrhœa. 4. Gonorrhœal peritonitis may exhibit various points of origin, so that it has been met with in the pelvic region, opposite the recto-vesical *cul-de-sac*, while at other times it arises at the internal orifice of the inguinal canal. 5. It may remain localized at the points where it has arisen and there undergo a cure; but it may also become generalized, or at least extend to a more or less considerable portion of the abdomen, pass on to suppuration, and terminate fatally. 6. Subperitoneal phlegmon has been observed in the lumbar fossa, at the lower part of the internal iliac region, and of the anterior wall of the abdomen. It may terminate either in resolution or suppuration, but its influence is less mischievous than that of peritonitis. 7. When a subperitoneal abscess has formed, it should be opened as soon as possible. An energetic antiphlogistic treatment, the prolonged application of ice, and preventive *débridement* may arrest the development of the phlegmon and prevent its passing into suppuration.

Gould (89) considers that stricture is more common in the bulbous portion of the urethra, because the discharge in a gonorrhœal inflammation will remain longer in that part of the urethra by reason of its horizontal position, and hence keep up irritation. In other portions of the canal, gravity helps to carry the discharge forward. If anterior to the bulb, it passes towards the meatus; if posterior to the bulb, towards the horizontal portion, which is limited in front by the penoscrotal bend.

Fauconnier (90) had the opportunity of examining carefully the urethra of a patient who died from erysipelas of the face while having a chronic gonorrhœa. The disease of the urethra was limited to the bulb, the membranous and prostatic portions were normal, as was also the anterior part of the canal. The mucous membrane of the bulb was slightly ulcerated and granular, and microscopically the granular spots were found rich in cells without any trace of fibrous interstitial substance. No appearance of a commencing stricture was observed.

Hardy (91) states that in gonorrhœal rheumatism the pains are sometimes very slight, and only manifested on moving; but in other cases they are extremely severe, and persist even during repose. There is a marked doughy tumefaction of the joints invaded, the amount of effusion being sometimes enormous, occasionally simulating a true hydrarthrosis. The erythematous redness of ordinary acute rheumatism is rare in this variety. It seems to have an especial predilection for the knee, after which come in order of frequency the wrist, ankle, shoulder, the fingers and toes, and especially the tarsus and metatarsus. But it is not always confined to the articulations, and for that reason it is preferable that it should be called rheumatism rather than arthritis, which has been proposed. Sometimes it is developed in the sheaths of tendons, at others in the tendinous bursæ, and more rarely in the sciatic nerve. Sometimes it occurs on one side and then on the other, and at others on both sides at once. While it is occupying these parts, various accidents are often met with in the eye, as intense conjunctivitis with suppuration, or keratitis accompanied by iritis—phenomena analogous to those observed in this organ during ordinary rheumatism. The number of joints affected differs from what is observed in febrile rheumatism; for while this last has a great tendency to invade several joints, and sometimes the whole of them, it is rare in gonorrhœal rheumatism for more than one or two, and sometimes three or four joints to suffer, and especially to find one after the other becoming affected, as is the rule in acute rheumatism. It is usually also apyretic, and if there is a little fever at first, this only lasts two or three days. So, also, the secretion of sweat is either absent or insignificant, and the changes in the urine, due to the preponderance of the urates and urea, met with in ordinary rheumatism, are absent. Finally, in this variety there are not the complications of heart disease; while, as a general rule, after lasting weeks or months, a cure results; but it sometimes gives rise to a true hydrarthrosis or a white swelling, with anchylosis. For the production of this affection not only is the existence of gonorrhœa essential, but there must also be a special predisposition which is not a tendency to the rheumatic diathesis. If the subjects of this disease be interrogated, it will be found that, independently of the blennorrhagia, their joints remain perfectly free, and they are nowise liable to contract muscular or articular pains on exposure to cold. Sometimes this rheumatism will appear at the very commencement of the urethral discharge, and sometimes only one, two, or three days later; and the rule which has been laid down, that the pains are severe in proportion to the abundance of the discharge, rests upon no foundation, for they are met with in the acute and subacute form of gonorrhœa, as in that which is manifested only by a slight discharge. It is not rare, when the articular pains appear, to find the gonorrhœa suddenly stopping, to return when they have been relieved. It would seem that a true metastasis takes place, the morbific material being transported from one place to another. But this phenomenon is far from being constant, and what is usually observed is that on the occurrence of the rheumatism there is only a certain amount of diminution of the discharge. Blenorrhagic rheumatism being, in fact, a local affection, and not being accompanied by general symptoms, measures which are purely local are those which alone succeed. Thus, at the onset, we should have recourse to application of leeches, dry cupping, and cataplasms, and, if the affection threatens to be prolonged, to blisters. At a later period, if it tends to a chronic condition, we may employ baths, douches, and mineral springs—the different means, in fact, that are used to combat chronic rheumatism.

Pirocchi (92) states that he has used dilute tincture of tayuya (ten parts in thirty or forty of water) as a local application in phagedænic and scrofulous ulcers, and in blennorrhagia. The ulcers became modified very soon after the application, twice or thrice in the day, of charpie steeped in tincture of tayuya; while in two cases of blennorrhagia the daily injection of the same remedy did not produce a favorable result. The author hence regards tincture of tayuya as a valuable topical remedy, capable, perhaps, of competing with the actual cautery in the treatment of phagedænic sores. He thinks that, being a tonic and astringent, it reduces suppuration in the soft parts, stimulates granulation, and facilitates cicatrization. As regards gonorrhœa, he does not consider tayuya superior to the balsams and other remedies ordinarily used.

(93) Gurjun balsam, in place of copaiba, has been prescribed with success for gonorrhœa at some of the hospitals of Paris. The following is Vidal's formula, as used at the Hospital Saint-Louis:—

Gurjun balsam, 4 grammes (1 drachm); gum, 4 grammes (1 drachm);

infusion of star anise, 40 grammes (10 drachms). To be divided into two doses, and taken immediately before meals.

Mauriac gives a larger dose: his formula at the Hôpital du Midi is as follows:—

Gurjun balsam, 16 grammes (4 drachms); gum, 10 grammes (2½ drachms); syrup of gum, 30 grammes (7½ drachms); mint water, 50 grammes (12½ drachms). To be divided into three parts, and taken during the day.

M. *Deval*, who watched the effects of the remedy in Vidal's service, recommends the former prescription, considering *Mauriac's* to be too powerful.

Gurjun balsam is cheaper than copaiba; it is also said to act more rapidly, and to have no disagreeable effect on the breath.

Gross (95), not accepting a cure of stricture of the urethra as possible by dilatation, advocates internal urethrotomy with the section carried up to the normal size of the urethra which is shown by *Otis's*, *Weir's*, and his own measurements to be an average corresponding to thirty-two of the French scale. He further believes that the size of the meatus is a guide to the size of the spongy urethra, the latter portion of the urethral canal being about eight mm. larger than the orifice.

Observing the rarity of orchitis (epididymitis) in connection with stricture, and its promptness when it occurs after operations involving the prostate, *Déspres* (96) does not concur in the theory that the testicular inflammation takes place by propagation along the vas deferens, but believes, especially in those cases where relapses and recurrences take place, that it is due to retention of spermatic fluid in the testicle, most probably from swelling or temporary occlusion of the ejaculatory ducts. He further states that blennorrhagic epididymitis is in proportion to the functional activity of the testes.

Terrillon (97). This author divides rupture of the urethra into three classes:

1st. Where it involves only the interstitial tissue, *i. e.*, the corpus cavernosum, the urethra being intact or at most only fissured.

2d. Those in which the urethra is alone involved.

3d. Those wherein there is laceration of the mucous membrane of the urethra, of the corpus spongiosum, and of the sheath of the penis.

The interesting fact is revealed by this observer, that it is extremely rare to find ruptures occurring posterior to the anterior layer of the triangular ligament. The treatment for the lighter cases, where there is no difficulty in micturition or only slight hemorrhage from the urethra, is to await the result of the case, but in the lesions of the third class, and often in those of the second class, it is desirable to proceed at once to an external perineal urethrotomy, as practised by *Desault* in 1805, and formulated by *Reybard*, in 1853. If, in a case beginning as a light one, retention should come on, it is better to relieve the symptom by aspiration rather than by the catheter. But in every such case, any perineal swelling should be an indication for a free incision.

Gay (100) has published what has long been practised by surgeons in overcoming the resistance offered by a tight stricture. He places the patient in a standing position, and gets him to urinate; while he is straining and while the urine is flowing, a fine bougie is to be introduced, when this manœuvre will often succeed in carrying the instrument through the stricture into the bladder.

Hanot (104) gives an account of a case of orchitis which occurred during the course of an attack of typhoid fever. The man, æt. 21, was admitted on August 19th, 1878. On August 25th (the sixteenth day of the fever), the patient complained of violent pain in the right groin and testicle, which had come on during the preceding night, and had prevented sleep. On examination, the scrotum was seen to be slightly tense and reddened ; the right testis somewhat swollen, harder than the left, and painful on pressure ; the epididymis was intact and painless, and the cord unaffected. There was no fluid in the tunica vaginalis. None of the ordinary causes of orchitis could be made out. On August 26th, the testis was as before, but the pain was less. On the 27th, the testis had slightly diminished in size, and the scrotum had become normal. On September 3d, all traces of the orchitis had disappeared. On the 14th, the patient was convalescent from typhoid fever, but the right testis had manifestly decreased in volume.

Some particulars of three other cases of affection of the testis and epididymis during typhoid are also given by Dr. *Hanot*. These cases occurred in the Hôpital Cochin, under the care of Dr. *Bucquoy*, in 1872 and 1873.

Esmarch (109) says of spasm of the urethra, which has been much discussed of late, that it is in his observation quite a frequent cause of retention of urine. The symptoms detailed are those given by *Delefosse*, *Caudmant*, and others to the *contracture du col de la vessie*. It is rarely idiopathic and may vary in severity from a case having only slight symptoms of dysuria to one associated with complete retention of urine. In the diagnosis, a large metal instrument is to be used, for, if a small one is employed, the spasm is often rendered worse by its point. The sensation sometimes felt when the instrument passes through the membranous portion of the urethra in a state of spasm is, as if it were passing through a rubber tube. This is, however, not felt if an anæsthetic is used. Sometimes it is difficult to introduce the catheter into the bladder, but it is never absolutely impossible in experienced hands. *Esmarch* also advises, when using the catheter or sound, to draw the penis strongly upwards, so as to obliterate as much as possible the bulbous dilatation of the urethra, and to neutralize the associated contraction of the sphincter ani, which, in its turn, tends through the perineal centre to pull the bulb further downwards. By this manœuvre the end of the instrument is carried more directly to the tightly-closed opening in the triangular ligament.

A sensitive hand can easily distinguish between a spasmodic and organic stricture, for the characteristic symptom of organic stricture, the 'bite' or grasping of the bougie, is wanting. *Dittel* is quoted as giving diabetes with $\frac{7}{10}$ per cent of sugar as a cause of spasmodic stricture. *E.* says further that anæsthetics always allow the bladder to be entered, corroborating the experience of all observers save *Otis* (108), who sets forth the contrary, with the additional statement that the bite given to the instrument in a spasmodic stricture cannot be distinguished from that of an organic stricture. *E.* relies on the frequent use of large sounds left in the urethra several minutes, with cold douches to perineum, and the exhibition of alkalies, bromide of potassium, camphor, etc.

Sand's (107) views, with those of *Reliquet* (105) and *Spire* (103), in the main agree with those of *Esmarch.* He presents also some collected cases, showing fatal results following the division of strictures of large calibre in supposed impassable spasmodic strictures.

Billroth (112) speaks of the mortality of dilatation of tight strictures in his hospital practice: 75 cases, 7 deaths, or 9.9 per cent, and looks with favor on internal urethrotomy. His dictum as to a *cure* of stricture is as follows: "I must, however, emphasize the fact that I regard internal urethrotomy and rapid dilatation (*Thompson's*) only as a means of hastening the cure, being of opinion that long-continued after-treatment by bougies is a necessity, so that neither these methods nor that of external urethrotomy are 'radical' in their cure."

Zeissl (114) believes chronic prostatitis to be the usual cause of gleet. In fact, he asserts that chronic gonorrhœa never exists without inflammation and some (not always discoverable by palpation) enlargement of the prostate. He also thinks this condition leads to permanent hypertrophy of the prostate.

Grünfeld (116) has by practice with the endoscope been able to recognize the colliculus. For this purpose he uses a straight large endoscope, and the view is best obtained in withdrawing the instrument. The colliculus is then seen as a dark-red horse-shoe like stripe, with the convexity upwards, from whose centre a swollen mass appears, strongly in contrast by its bright-red color with the surrounding mucous membrane. He has not yet been able to detect the mouths of the ejaculatory ducts. *Gschirhakl* has not, however, been able to distinguish blood-vessels as *Grünfeld* claims to have done.

Grégory (117), in a comprehensive thesis, has aimed to develop the idea that external urethrotomy is not only a safer method of treating strictures than any of the other operative (*i. e.*, cutting) procedures, but that it is also more likely to cure a stricture radically; and that relapses are not so frequent after its performance as they are after internal urethrotomy. He gives 915 cases of internal urethrotomy, with 46 deaths, a mortality of 5%, but there should be deducted from this figure the 116 cases, without a death, furnished by *Berkeley Hill* and *Otis*, as the operation in these was resorted to for strictures of large calibre, and only cloud the consideration of the case. Omitting these, we have 800 cases with 46 deaths, or 5.66% mortality. As a contrast to this, *Grégory* gives 992 external urethrotomies with 88 deaths, or a mortality of 8.87%. But, wrongly it seems, he throws out of the list in each operation the deaths which resulted from advanced renal lesions, and satisfactorily places them so that the number after internal urethrotomy is reduced to 39, giving a death rate of 4.87%, and after external urethrotomy to 30, and a death rate of 3.02% in lieu of 8.87%.

As to the return of stricture after the external section, *Grégory* presents 24 cases out of 61 in which a return had not occurred after a period of more than a year had elapsed. However, it is not clearly mentioned whether, in all of these cases, a sound had not been passed from time to time by the patient. In 46 cases where this point was observed, 9 complete cures were recognized, a result certainly encouraging.

Sabourin (122) investigated this subject as well as *Dartigues* and *Nicaise*, and believes that the retention of urine after a traumatism elsewhere is due rather to paralysis of the expulsory muscles of the bladder than to spasm, inasmuch as when the catheter is introduced, in the majority of cases, the urine will be noticed to flow sluggishly, and with oscillations corresponding with the movements of respiration. Spasm is more likely to be the cause in connection with strangulated hernia, and operations or inflammations about the rectum and anus, but for distant injuries, such as fractures, etc., the cause is more likely to be the atony or temporary paralysis of the bladder.

Mastin (123) had a case of urethral stenosis in a man of forty-six years due to gonorrhœal inflammation treated with strong injections of nitrate of silver, which was situated at seven and one-quarter inches from the meatus urinarius without stretching the urethra, and for which external perineal urethrotomy was performed. After the membranous urethra had been opened at the apex of the prostate, a small probe was passed through the stricture, which was thus positively determined to be in the prostatic portion of the canal. The stricture was then divided; no evidence either per rectum or by the wound existed of prostatic hypertrophy or of other disease save the stricture. This case is more difficult to controvert than those reported by *Leroy*, *Ricord*, and *Walsh*, which have been somewhat doubted by *Sir Henry Thompson*.

Dugas (124) uses, in the treatment of orchitis, a bandage of bleached sheeting one inch wide and four yards long, which is imbued with starch before being applied. The bandage is to be renewed night and morning.

Terrillon and *Schwartz* (125) state, in reference to the hydrocele usually met with in attacks of epididymitis, that it is rare to have this complication unless the epididymitis is of gonorrhœal origin. The scrotum is usually the last in the extension of the inflammation.

They quote from *Sigmund* 1342 cases, of which in but 61 was there epididymitis only; in the rest it was conjoined with vaginalitis or funiculitis, and from *Zapata* 152 cases in which vaginalitis occurred 62 times. These observers also made a number of injections of irritants into the vas deferens and also into the substance of the testis in dogs, and showed thus that the transition of inflammation from the cord to the tunica vaginalis was due to their anatomical connection by means of loose connective tissue, and that, in orchitis, the serous membrane was not involved by reason of the presence of the tunica albuginea.

White (127) describes a thin hard-rubber case which, inclosing the swollen testis, can readily be made smaller by lacing together the separated edges on its anterior surface.

VIII. IMPOTENCE AND STERILITY IN THE MALE.

BIBLIOGRAPHY.

128. Gross, S. W.: Organic Stricture of the Urethra from Masturbation, also Sexual Debility and Impotence from Stricture, etc. Phila. Med. and Surg. Reporter, May 5th, 1877.—129. Hardy: Impotence an Early Sign of Diabetes. Gaz. des hôp., May 22d, 1877.—130. Winternitz, Dr.: Psychrophor or the Cold Sound. Berl. klin. Wochens., July 9th, 1877.—131. Lerreboullet, M.: Atrophy of Testes following Mumps. Med. Times and Gaz., Sept. 1st, 1877.—132. Ultzmann, R.: Ueber männliche Sterilität. Wien. med. Presse, Nos. 1, 3, 1878.—133. Ultzmann, R.: Die endoscopische Untersuchung des Samenhügels. Wien. med. Blät., Nos. 38, 39, 1878.—134. Bryant, T.: Strumous Disease of the Vas Deferens Simulating Iliac Abscess. Med. Times and Gaz., June 29th, 1878.—135. Weiss, W.: Beiträge zu den angeborenen Verengerungen der männlichen Harnröhre. Jahresbericht von Virchow und Hirsch, 1878.—136. Berg, R.: Om Aspermatozi og Aspermatisme. Hospitalstidende, p. 721, 1878. Jahresbericht, 1878, Virchow und Hirsch. —137. Caldwell, J. J.: Experiences in the new drug Damiana (Turnera Aphrodisiaca). St. Louis Med. and Surg. Journ., June, 1878.—138. Laveran, Dr.: Mumps and Parotidean Orchitis. Medical Times and Gaz., July 20th, 1878. —139. Gross, S. W.: Prostatorrhœa and Stricture of the Urethra. Phil. M. Times, Sept. 28th, 1878.—140. Reliquet et Cadiat: Sympexions volumineux d'une vésicule séminale chez un sujet sain. Bull. Acad. de Méd., Sept. 17th, 1878.—141. Trélât: Kystes du cordon spermatique. Le Progrès Méd., Nov. 23d, 1878. 142. Gérard: Observation de féminisme coincidant avec une atrophie complète des deux testicules. Rec. de mem. de méd. chir. et pharm. militaires, p. 187, 1878. —143. Un pénis en détresse. Lyon Médical, XXIX., p. 315, 1878.—144. Ferrant, Ch.: L'inflammation des vésicules séminales comme cause de pollutions nocturnes. Rev. de thérap. méd. chir., p. 622, 1878.—145. Salzer: Long Continued Priapism accompanying Leukæmia. Lond. Med. Record, Sept. 6th, 1879. —146. Annandale, T.: Transplantation of Testis from the Perineum to the Scrotum. Brit. Med. Journ., p. 7, 1879.—147. Mœbius, P. I.: Ueber die Behandlung der Spermatorrhœa. Memorab. Heilbrun, XXIV., p. 545, 1879.—148. Zangrilli, A.: Il bromero di potassio e le coca nelle impotenza virile. Gazz. med. di Roma, V., p. 73, 1879.—149. Adams, I. E.: Transition of Testis into Perineum. Brit. Med. Journ., I., 227, 1879.—150. Greenfield: Spermatorrhœa and Rheumatism. Trans. Clin. Soc., March 1st, 1879.—151. Grünfeld, I.: Endoscopische Untersuchung des Colliculus seminalis. Wien. med. Presse, XX., 781, 1879.—152. Caldwell, J. J.: Impotence and Sterility. Treatment by Damiana and Electricity. Virginia Med. Monthly, VI., 436, 1879.—153. Reliquet: Coliques Spermatiques. Gaz. des hôp., 891, 1879.—154. Bartholow, Roberts: Spermatorrhœa. Fourth Ed., 1879.—155. Heinemann, H. N.: Double Cystic Development of the Vesiculæ Seminales. N. Y. Med. Jour., March, 1880.—160. Ranney, A. L.: Atrophy of Penis following Internal Urethrotomy. N. Y. Med. Journ., May, 1880.

—161. Latulonne: Des troubles mentaux dans les maladies des voies urinaires. Pau médic., 163, 1878.—162. Wilkes, A. B.: Reflex Neuralgia of the Genito-Urinary Tract Cured by Quinine. Southern Pract., I., 96, 1879.—163. Knott, L. D.: Paralysis from an Elongated Prepuce Cured by Circumcision. Louisville Med. News, Feb. 22d, 1879.—164. Stevens, C. B.: Sexual Neurotic Conditions. The Physician and Surgeon, Michigan, II., 1, 1879.—165. Caldwell, J. J.: Vaso-motors, Vaso-Dilators, and Vaso-Constrictors of the Genito-Urinary Organs. St. Louis Eclectic Medical Journ., VI., 529, 1879.—166. Westmoreland, R. W.: Reflex Nervous Troubles from Genito-Urinary Irritation. Atlanta Med. and Surg. Journ., XVII., 387, 1879.—167. Le Dentu: Paraplegia Urinaire. Journ. de méd. et chir. prat., L., 304, 1879.—168. Roberts, C. H.: Paralysis from Spermatorrhœa. Canada Medical Record, VII., 253, 1879.—169. Ultzmann, R.: Ueber Neuropathien des männlichen Harn- und Geschlechts-Apparatus. Wiener Klinik, V., 119, 1879.—170. Beard, G. M.: Nervous Diseases Connected with the Male Genital Function. Medical Record, Dec., 1879, May 8th, 1880.—171. Vance, R. A.: Constriction of the Urethral Orifice in Adult Male, the changes it induces, etc. Indiana Med. Reports, I., 10, 1880.

Gross (128) claims that strictures not infrequently result from masturbation, and cites nineteen instances which he had encountered of this origin in a total of one hundred and thirty-eight cases in which the cause of the urethral stenosis had been ascertained. Of these nineteen cases, he found the stricture to be of large calibre, single, and situate at an average depth of five and seven-tenths inches from the meatus urinarius. As there was no history in these cases of any purulent urethritis, the suspicion cannot be avoided that Dr. *Gross* has been misled by the bougie à boule catching in the anterior layer of the triangular ligament. Inasmuch as Dr. *G.* proposes urethrotomy for the relief of these strictures, it is to be hoped that the subject will be fully considered before such a useless risk is run.

The same author further declares (139), concerning prostatorrhœa, that in this disease no spermatozoa are to be found, and that such discharges can be distinguished by the presence of the so-called prostatic concentric calculi. For the treatment of this affection, he relies upon his dilating sound (see N. Y. *Med. Record*, June 15th, 1878) and the use of solid nitrate of silver applied by Lallemand's porte caustique.

Hardy (129), in a lecture on diabetes, remarks that an early sign of approaching diabetes is genital impotence, and that where in individuals this symptom presents itself without coinciding disease, especially of the spinal cord, diabetes will be found to be the cause.

Winternitz (130), of Vienna, has designed an instrument, by means of which he secures the advantages of mechanical irritation of the urethral mucous membrane by the metallic sound, in combination with the anæsthetic and tonic influence of cold. It consists of a double-current catheter without eyes, the two canals communicating with one another near the point of the instrument. The instrument is introduced into the urethra until its point has passed the prostatic portion, and it is then attached by India-rubber tubing to a reservoir containing water at the desired temperature. On turning a stop-cock, the water flows into one canal and out through the other, whence it is conducted away by another piece of tubing. In this way the caput gallinaginis and the entire urethral mucous membrane are exposed to the mechanical action of pressure, and to the sedative action of cold. The success obtained by *Winternitz*, by the use of this instrument, was so encouraging from the very beginning that he has employed it constantly for over a year.

He has treated with it twenty-two cases of pollution. Of these two did not return after the first application; one was improved at first, but soon became as bad as before, and the treatment was discontinued after the cold sound had been used sixty-five times; twelve are still under observation, and have been so much improved by the treatment, that the pollutions occur very rarely, and the secondary symptoms, hypochondria, etc., have entirely disappeared. In three cases the improvement was marked, when the patients withdrew from observation; in two others the pollutions became less frequent, but the secondary symptoms remained unchanged. The two remaining cases are described in detail. In one, the patient was a Russian officer, forty-six years of age, and the affection was due to excessive venery. The pollutions occurred regularly in the night after coitus, and recurred two or three times a week, when the patient was continent. The cold sound was used daily for ten minutes with water at 59° F.; during its employment the patient experienced a sensation of pleasant coolness, and the relaxed scrotum contracted energetically. Some difficulty was experienced in removing the instrument. During the four weeks that the treatment was continued, there was only one pollution. The erections became more complete. In the second case the pollutions were frequent, and there were symptoms of excessive spinal irritation. The first introduction of the instrument caused great pain, and brought on an hysterical fit, but these symptoms disappeared after the water (59° F.) had flowed through the sound for five minutes. The treatment was continued daily for three weeks, when the patient was discharged cured. He had not had a single pollution from the time the treatment was begun.

At the first sitting *Winternitz* sometimes uses water at a temperature of 64° or even 66° F., and at a later period sometimes goes as low as 54½° F. Besides the above, he has treated nine cases of spermatorrhœa with this cold sound. In four of these cases he obtained very favorable results; two cases were very markedly improved, while in the other three the treatment was without special results. In the cases of spermatorrhœa as well as in those of pollution, in which the treatment proved successful, general relaxation of the genitals and loss of muscular tone in the scrotum were marked symptoms. The cold sound was also used in five cases of too rapid ejaculation during coitus, and in two cases of obstinate chronic gonorrhœa. In the former its use was followed by at least temporary improvement, and both of the latter, one of which had lasted three years and the other six months, were cured.

Lereboullet (131) recently brought forward at the Société Médicale des Hôpitaux a "Contribution to the Study of Atrophy of the Testes and Hypertrophy of the Mammary Gland in Consequence of Certain

Kinds of Orchitis." His remarks were founded on a case, of which the following is a *résumé:* A young man, aged 22, of robust health, well made, and possessed of all the characters, physical as well as physiological, of virility, was attacked with mumps. The disease seemed mild; at the commencement there was no febrile action and no complication. At the end of four days, although the parotid swelling had not disappeared, a double orchitis came on, so that within two days these glands were swollen to triple their normal size. The pain, however, was not great; *the epididymis was normal.* The disease evolved rather rapidly. When admitted into the Val-de-Grâce, the patient still presented peri-parotid swelling, and yet the atrophy of the testicle was well advanced. In about three weeks these glands were no larger than a haricôt bean. At the same time that the testes were undergoing this atrophy, and the sexual power and desire were disappearing, the mammary glands, which up to that time had been absolutely normal as for a young man of this age, began slowly but progressively to develop. But now, although there are no other external signs of femininism, yet one is distinctly struck by the development of the breasts; to the touch they are lobulated and hypertrophied, and do not at all feel like mere fat; they are increasing in size daily; the nipple undergoes erection after any excitation which is prolonged. There is an absence of beard, although the pubic hair continues. The penis has a normal development, but there is an absolute loss of the genetic sense. This case goes to show that an orchitis has supervened before the complete disappearance of the parotid troubles, and that it complicates a disease apparently mild in its type. This is not by any means an exceptional case or an exceptional complication. Many similar cases are recorded, especially in France. Some authors, and among them M. *Juloux,* believes that the atrophy, though it comes on slowly, nevertheless comes on surely; and M. *Laurens* reported, out of thirty-two cases, sixteen of atrophy of the testes, and nine times impotence. M. *Lereboullet,* however, cannot accept these conclusions; he believes rather that the testicles may become modified, both as to volume and consistence, without there being any real progressive or persistent atrophy of these organs. He thinks that army surgeons, seeing the frequency of epidemics of this disease (mumps) would meet with more cases among old soldiers than they actually do, if the atrophy occurred at all frequently and persistently; and hence he concludes that the atrophy is only partial, and that it does not remain permanent. Atrophy of the testicles also occurs after traumatic orchitis as the result of excessive masturbation, and, though very rarely indeed, after gonorrhœal orchitis or syphilitic orchitis.

Ultzmann (132) gives, in his article, an example of permanent aspermatism, which is very rarely met with. It was in a man twenty-four years of age, strongly built, who had never had a seminal ejaculation. He had occasional erections, never found any special pleasure in coitus, and never had in it an ejaculation. He had never, to his knowledge, had a pollution at night.

The genitals were normal, both testes and penis of the proper size, as was the prostate. A sound passed into the bladder without difficulty. The urine was daily examined; it was normal, and the sediment never contained spermatozoa. The patient had never had either gonorrhœa or syphilis. Faradization of the testes was tried, to incite the formation of spermatozoa, without effect. In spite of repeated coitus and sexual excitement, the patient could produce no semen, and he left Vienna after a fruitless treatment lasting several weeks.

Ultzmann further states that azoospermatism is much more common than aspermatism. He gives three varieties of seminal fluid which are destitute of spermatozoa. 1st, The catarrhal or purulent sperm. This presents a considerable white sediment, with much epithelium, pus, and blood, due to an inflammation proper of the vesiculæ seminales. 2d, The watery sperm. In this the spermatic crystals are well developed, which, in the normal secretion, are only partially so, or are absent. Cylindrical epithelium and molecular detritus in motion are also found. 3d, The colloid sperm. No crystals are perceived in this form of the seminal fluid, but large masses of colloid epithelium and stratified globules. The secretion is thick and white. This form is encountered in cases of obliteration of both efferent ducts. The watery and colloid spermatic fluids are associated with absolute and permanent sterility.

Bryant (134) detailed a case which at first sight resembled an incarcerated hernia. No history of gonorrhœa was present. A swelling existed in the scrotum three inches in length, and without any impulse on coughing. On being opened, two and a half ounces of pus were discharged. Patient died; the autopsy revealed caries of the vertebra. The cord showed, from the bladder to the testes, tubercular infiltration with abscesses separate from the spinal affection. The testis and prostate were also tuberculous.

Weiss (135) made an autopsy of a thirteen-year-old boy who had had peri-urethral abscess and urinary infiltration. There was found at the end of the first third of the urethra, in its lower wall, a semilunar fold of mucous membrane with its free border looking towards the bladder. When this flap was lifted up, it was three millimetres deep. A catheter passed inwards could not dectect it, though a bougie à boule was caught by it when withdrawn. The bladder was thickened and kidneys dilated. The patient, during life, resorted to pulling manipulations to press forwards the urine.

Berg (136) is inclined to believe, though he admits that no proof has yet been obtained by a post-mortem examination, that distortions or diseases affecting the caput gallinaginis must often be a cause of aspermatism. He presents an interesting case of this form of sterility, viz.: a young man who, in his first coitus, had the usual ejaculation, but soon afterwards failed in this respect, though he often subsequently felt an unpleasant sensation of pressure in the deeper parts of the urethra. This continued for five years. Four months before he was examined he had had two copious nocturnal pollutions. He was married, but was childless. The inspection of the external genitals, urethra, bladder, etc., revealed nothing abnormal. During a certain coitus with a condom, a sensation of bursting was noticed, and the patient evacuated a thick fluid, which contained a great number of spermatozoa and seminal cells, also some quite hard lumps of yellow color, consisting of closely-packed spermatozoa, epithelium, fat-cells of yellow-brown color, and lastly, various-sized sympexions. Subsequent coitus, at times, was devoid of emissions. The woman became pregnant.

Urethritis, according to this observer, will cause closure of the ejaculatory ducts, and, in such instances, a species of spermatic colic may be noticed, the patient experiencing twitches along the spermatic cord, with sensations as if an emission were about to occur. Sometimes there is a feeling of bursting and after that a blood-stained pollution. Another case is given where temporary aspermatism, *i. e.*, aspermatism with nocturnal pollutions, was due to a phimosis, for, after circumcision, the genital function was completely re-established. This form of aspermatism—temporary—may be caused by excessive excitation of the parts and sometimes by the imbibition of a considerable amount of liquor. This is somewhat like a case related by *Roubaud*. *Berg* finally gives an instance where coitus with emission with one woman was impossible, but with others it was normal.

The paper of *Caldwell* (137) contains nothing new on sterility. In impotence he has had good results from the administration of the fluid extract of damiana ʒ ij., three or four times a day, sometimes conjoined with the use of the constant electrical current, as advised by *Benedict*.

Dr. *Laveran* (138), of the Val-de-Grâce, recently read a communication to the Hospital Medical Society, in answer to a question asked by Dr. *Besnier* in one of his sanitary reports, as to the frequency of the occurrence of parotidean orchitis and the subsequent wasting of the testicle, and as to the prophylactic measures likely to prevent these accidents. It is impossible to reply exactly with regard to the frequency of the occurrence of mumps in the military hospitals, as the statistics of these establishments do not include it; but it may be stated generally that small epidemics of the affection often occur. In 432 cases of mumps observed among soldiers in very different localities, there were met with 156 examples of single or double orchitis, so that this complication may be said to occur in two out of five cases in adults, although it is very rarely met with in epidemics occurring in schools. The orchitis usually occurs from the sixth to the eighth day after the appearance of the mumps, just as the swelling of the parotid is beginning to disperse. Attributing this to metastasis is out of the question, for the mumps does not subside any more rapidly in the cases in which orchitis occurs than in others, while it may arise spontaneously, unpreceded by any affection of the parotid. Although in some epidemics double orchitis has been observed oftener than single, as a general rule one case of double is met with for five or six cases of single. The degree of inflammation of the testicle is as variable as is that of the parotid, chiefly affecting the substance of the testicle itself, the epididymis suffering only in a less degree. By the fourth day the testis has increased two or three times in size, and is very hard and very tender to the touch. Resolution soon takes place, and there is in general no effusion into the tunica vaginalis. Unfortunately, however, the disease frequently terminates in atrophy, a condition which has often been overlooked in civil practice, in which the patients are not so long under observation as soldiers; and the atrophy does not take place sometimes until weeks or months after. In 111 cases of parotidean orchitis, atrophy occurred in 73, *i. e.*, in 7 out of 10 cases. When atrophy affects both testicles, which is rare, complete impotence ensues; and when only one testis is affected a considerable diminution of virile power occurs. Sometimes the atrophy is arrested, and the testis recovers its normal size and consistency; but in general it persists, and not infrequently the other testis undergoes a compensatory hypertrophy.

With respect to prevention, *Laveran* believes the contagiousness of mumps to be amply demonstrated, and that the disease offers the same specific characters as the eruptive fevers. Isolation is, therefore, indicated, at least as regards adults. As soon as soldiers are affected with mumps, they should be sent to hospital, and not allowed to mingle with the other patients there. As to preventing the disease localizing in the testis, no means are known, all that can be done being to recommend rest.

Reliquet and *Cadiat* (140) submitted a specimen taken from a recently executed criminal of a vesicula seminalis, more or less filled with sympexions, as described by *Robin* in his *Traité des Humeurs*, p. 443. No carbonate of lime was found in them, only fatty granules, hematosine, and globules.

Salzer (145), of Worms, reports the case of a man, forty-six years of age, who suffered for seven weeks from persistent priapism. He had previously suffered from intermittent fever, but was at this time in apparent good health. One morning he was awakened by an intensely painful erection of the penis, that proved utterly rebellious to treatment. Leeches, warm fomentations, chloral hydrate, and even chloroform narcosis were tried in turn, but all without success. The urine was passed with difficulty, usually in short jets, and most readily in the knee-elbow position. Physical examination revealed only *marked enlargement of the spleen*. Finally, after the penis had been kept for three weeks constantly enveloped in strongly camphorated narcotic poultices, opium and camphor being administered internally at the same time, the priapism gradually disappeared, having persisted fully seven weeks. During the week preceding this attack, the patient had had two attacks of priapism, one of which lasted only a few hours, and the other twenty-four hours. After the appearance of the priapism the patient rapidly lost strength and acquired a cachectic appearance, and the spleen progressively increased in size. Two months after the priapism disappeared there was complete loss of sexual power, and the patient died about eight months afterward. The blood was not examined microscopically, and an autopsy was not permitted.

Dr. *Salzer* collates from medical literature four other cases of priapism occurring in connection with leukæmia. Various theories have been brought forward to account for the priapism in these cases. *Klemme* ascribed it to extravasation of blood into the corpora cavernosa, and *Longuet* to impeded circulation in the smaller vessels, and the formation of thrombi, resulting from the altered condition of the blood, while *Neidhardt* thought that irritation of the nerves might possibly be the exciting cause. Dr. *Salzer* thinks that the rapid disappearance of the priapism in the two first attacks in the above case argues against the occurrence of an extravasation of blood. He believes that both the temporary and the persistent attacks of priapism were due to irritation of the nervi erigentes. It is well known that priapism may he produced both by peripheral and by central irritation of these nerves. As examples of the former, he adduces the erections accompanying inflammation of the urethra or of the neck of the bladder, swelling of the prostate, etc.; and, as examples of the latter, the erections of insane persons, or that follow injuries of the spinal cord. The priapism of leukæmia, he claims, differs from these varieties chiefly in its longer duration, and hence for its development some special cause must be sought. This may possibly be found either in the presence of anatomical changes in the nervi erigentes, or in pressure on them by swollen lumbar glands.

Greenfield (150), in an interesting discussion on gonorrhœal rheumatism, cited the case of a young man, twenty-five years of age, who had long been the subject of seminal discharges and of irritation about the prostatic urethra. In this patient all the joints of his hands and feet would become affected with rheumatic pains and swellings whenever the spermatorrhœa was excessive.

Reliquet (153), referring to a case reported by him in 1874, where sympexions shut up the ejaculatory duct of one side, causing great pain, sexual excitement, and erection, and in which relief was afforded by the introduction of a lithotrite, which opened or removed the obstruction from the urethral end of the duct, reports another case in which the same symptoms were produced, but in which some masking occurred from the paroxysmal signs of calculus presented in the history. The diagnosis at first made was tubercle of the prostate. The prostate, when examined by *Reliquet*, was found irregular, especially on the right side. This swelling was continuous with an enlargement of the seminal vesicle on the same side. While a sound was in the bladder, and a finger in the rectum, strong pressure was made by the latter, after which was discharged, per urethram, a grayish matter, in shape like vermicelli, and not readily crushed, which under microscopical examination by *Robin*, showed masses of spermatozoa and mucus with a few sympexions and epithelial scales. Subsequently, the patient himself expelled other similar masses, and the cure was effected by the aid of pressure once or twice repeated.

Heinemann (155) presented at the New York Society of German Physicians a specimen of double cystic development of the seminal vesicles. They were nearly treble their normal size. He was able to ar others like it, reported by *Pitha* and *Billroth*, *Englisch*, *eabody*. The apparent infrequency of similar cases he y to be due to the fact that a post-mortem examination ally include an inspection of the posterior surface of the

30) saw a case in which circumcision and internal urethro- performed some time previously and apparently for very symptoms. The penis was found to have atrophied to one , with abolition of sexual desire. Testicles normal. The is imperfect, as the locality of the urethrotomy and the ion of the urethra are not stated. The patient, moreover, y three hundred pounds, and it is possible that the absorp- is was only an apparent and not a real one.

DISEASES OF THE LIVER.

JAUNDICE.

CHOLECYSTOTOMY.

HYPERTROPHIC CIRRHOSIS.

BY

R. H. FITZ, M.D.,

OF BOSTON,

PROF. OF PATHOLOGICAL ANATOMY IN HARVARD UNIVERSITY.

JAUNDICE.

The evidence on which the occurrence of a hæmatogenous jaundice mainly rests is based upon the clinical observation of a yellowish discoloration of the skin, associated with the presence of bile-pigment in the urine, and the absence of bile-acids from the fluids of the body. The slow pulse and itching of the skin, which are usually present in jaundice of biliary origin, are lacking. Under such circumstances, open bile-ducts are likely to be found. Additional evidence has been sought for in the occurrence of jaundice after profuse hemorrhages, and in the results of the experiments of *Ponfick* and *Tarchanoff*. These observers found that, after the injection into the blood of solutions of blood-pigment, or of crystallized hæmoglobine, there subsequently appeared hæmoglobine in the urine, to be followed later by the presence of bilirubine in this fluid.

It seemed, therefore, fair to infer that, if a sufficient number of red blood-corpuscles were destroyed, a jaundice might result from the abundant presence of free blood-pigment in the circulation.

*Kunkel** objects to this inference, as there is likewise an increase of the bilirubine secreted in the bile, and it is possible that a limited catarrhal jaundice might be present in some part of the liver, in consequence of which the bilirubine makes its appearance in the urine, as observed in the experiments alluded to. He has found that a limited obstruction of bile-ducts may take place in the liver, and jaundice result, although bile is still flowing through the hepatic duct. This observation is also explanatory of those cases where the stools are dark-colored, although the individual is jaundiced.

It has been stated by *Poncet* that, in the jaundice following extensive hemorrhages, the pigment eliminated in the urine was like urobiline, and *Dreyfus-Brisac*† has endeavored to present the means of distinguishing clinically between such a form of jaundice and that resulting from the absorption of bile. In the former variety, the discoloration of the skin is rather dirty-yellow or pale-yellow, without the greenish tint which is common in hepatogenous jaundice. The feces are not clay-colored, and the urine is of an amber-yellow or brownish-yellow, without any greenish tint. The nitrous acid test of *Gmelin* produces no specific reaction, and merely a reddish-brown color results when nitric acid is added to the urine. Chloroform extracts from this secretion a brownish-red coloring matter, which becomes of a rose-red color when acted upon by nitric acid. There is neither itching nor a slow pulse.

---

* Virchow's Archiv, 1880, Vol. LXXIX., p. 455.

† Revue des Sciences Médicales, 1879, Vol. XIII., 1er fascicule.

Gerhardt[*] has recently corroborated these observations, and maintains that, in cases of jaundice, where the ordinary biliverdine reaction is not present, the dark color is usually due to the abundant presence of urobiline.

This urobiline-jaundice was first recognized as the result of internal hemorrhage, and the relation which urobilinuria presents to extravasated blood has been specially studied by *Kunkel.*[†] When an extensive hemorrhage has taken place, the blood-pigment is eventually removed and presumably eliminated. During this period of removal, the urobiline in the urine becomes very much increased. It is therefore likely that this increased secretion represents the result of the absorption of the extravasated blood. The urine in febrile affections likewise contains an increased quantity of urobiline, and the absorption of large quantities of blood is accompanied by fever. That the urobilinuria connected with hemorrhagic extravasation is not the result of the fever is to be inferred from the quantity of the pigment, which is far greater than that in simple febrile disturbances, and from the time of its appearance, which is not dependent on the course of the fever.

It is uncertain what may be the relation of urobiline to hæmoglobine. It is generally agreed that bile-pigment is derived from blood-pigment, and many observers regard the former as absolutely identical with hæmatoidine, that form of blood-pigment which results from extravasation. That urobiline may likewise be derived from hæmoglobine is not improbable, although the evidence is wanting which may confirm this view.

If the jaundice from extravasation is the result of the discoloration of the tissues produced by the dissolved and deposited coloring matter of the blood, hæmatoidine or bilirubine, it differs from a hepatogenous jaundice, and what has hitherto been regarded as a hæmatogenous jaundice, in the absence of bilirubine in the urine. There has been no analysis of the pigment present in the tissues in this form of jaundice, and it is therefore not apparent whether it consists of hæmatoidine, bilirubine, or of urobiline.

In the removal of bile-pigment from the tissues in hepatogenous jaundice, it is found that at first bile-pigment alone is present in the urine; later, both bilirubine and urobiline are present, while later still only urobiline is present.

The importance of the recognition of urobiline-jaundice is manifest when the frequent associations of jaundice with pneumonia, heart-disease, and hemorrhagic infarction of the lungs is remembered. And it is claimed that the presence of urobiline-jaundice may be regarded as evidence of internal hemorrhage.

---

[*] Allg. Med. Central-Zeit., 1878, No. 95, p. 1187.

[†] Loc. cit.

It is likewise suggested that in the jaundice of new-born children the icterus is to be considered at times as resulting from the hemorrhagic accidents of birth, as the cephalhæmatoma or some internal hemorrhage.

CHOLECYSTOTOMY.

The operation of opening the gall-bladder, cholecystotomy, has attracted a considerable degree of attention during the past two years, chiefly in consequence of the report of a case by Dr. *J. Marion Sims.*[*]

The earliest mention of such an operation dates back to the last century, and in a discussion which took place at a meeting of the London Clinical Society, it was stated by *Hulke*[†] that the whole question had been exhaustively treated in an early number of the *Mémoires de Chirurgie*, published about the year 1706. The feasibility of this operation for the removal of impacted gall-stones was urged by *Petit* in 1743, and has been suggested at intervals since that date, mainly by English writers.

The case reported by Dr. *Sims* was one of severe jaundice, and the diagnosis of a cyst connected with the liver had been made. The gravity of the symptoms urged Dr. *Sims*, who had been seeing the patient as consultant, to recommend opening the abdominal cavity, and the gall-bladder if necessary.

The incision through the abdominal walls was three inches in length, parallel with the linea alba, and was about three inches to the right of the umbilicus, being made over the most prominent part of the abdominal tumor. The latter was then emptied of its contents by means of an abdominal trocar, and was ascertained to be the gall-bladder, its relations to the liver being determined by passing the finger over the peritoneal surface. Some two inches of the gall-bladder were cut off, and sixty calculi removed from its cavity.

It was then determined to create a biliary fistula, and more of the gall-bladder was cut away, the free edge being united to the upper end of the abdominal wound. The patient lived eight days after the operation. Death was attributed to hemorrhage, in consequence of the poisonous effects of biliary salts, and no traces of peritonitis were found at the post-mortem examination. The edges of the gall-bladder were found to be firmly adherent to the abdominal wall. A probe could be passed from the remains of the gall-bladder through the common duct into the duodenum. The hepatic ducts were dilated. Sixteen gall-stones, the largest of the size of a pigeon's egg, and all stated to have been sacculated, were removed after death from the gall-bladder.

Although this case is the first reported among the recent contributions to the subject, Mr. *George Brown*[*] had already opened the abdominal cavity, a communication with the gall-bladder being soon established, and the patient was convalescing at the time Dr. *Sims* operated. In Mr. *Brown's* case, the patient had a tumor in the region of the liver, which was supposed to be an abscess, and was aspirated. There being no material relief, the abdomen was opened to the right of the umbilicus and over the tumor. When the peritoneum was cut through, it was found that the tumor was to the left of the umbilicus, the dulness and swelling near the incision being attributed to the inflammatory enlargement and adherence of the omentum. Some of the adhesions at the left of the wound were separated, but it was thought inexpedient to extend the operation, and the abdominal wound was closed with three sutures. Retching and vomiting took place in the night, and a yellowish fluid, which proved to be bile, oozed from the wound. This fluid continued to be discharged for three days, when it was replaced by fetid pus. The patient sat up eleven days after the operation, and walked out at the end of the month.

---

[*] British Medical Journal, 1878, June 8th, p. 811.

[†] British Medical Journal, 1879, May 31st, p. 819.

Mr. *Bryant*[†] reported the history of a case of cholecystotomy before the London Clinical Society. The patient came under his charge with two sinuses in the abdominal wall, of three years' duration, which communicated with an abscess that had begun to form two years before the sinuses appeared. On opening these sinuses, pus first made its appearance, then bile, and an exploratory operation was performed, which led to the removal of a gall-stone lying at the depth of two inches. Bile continued to flow from the opening for two weeks, and definite healing took place in four months.

The next case recorded is that reported at a meeting of the Royal Medical and Chirurgical Society by *Lawson Tait*.[‡] The patient had a heart-shaped tumor lying over the right kidney, with regard to the nature of which no decided diagnosis was made. It was firm, elastic, without fluctuation, could be moved laterally, and was tender. An abdominal incision, four inches in length, was made along the linea alba, and the tumor was found to be the gall-bladder distended with a starchy-looking fluid. After opening the gall-bladder, an impacted calculus was removed from the cystic duct. The wound in the gall-bladder was stitched to the upper end of the wound in the abdominal wall by a continuous suture, a biliary fistula being established. Bile flowed through the opening for eleven days, and the wound healed six days after the bile ceased to flow.

Dr. *W. W. Keen*[§] has reported a case of cholecystotomy which terminated fatally thirty-six hours after the operation. There was no peritonitis, and death was attributed to shock, secondary hemorrhage, and a generally deteriorated condition. The hepatic ducts were found to be dilated, the cystic duct not being enlarged. The condition of the common duct was not ascertained.

---

[*] British Medical Journal, 1878, Dec. 21st, p. 916.

[†] British Medical Journal, 1879, May 31st, p. 819.

[‡] The Lancet, 1879, Nov. 15th, p. 729.

[§] The American Journal of the Medical Sciences, 1880, Jan., p. 134.

Although merely some of the prominent points occurring in the several reported cases are here presented, it is evident that the operation of cholecystotomy has thus far met with but little actual success. The case operated on by Mr. *Brown* is open to doubt concerning its actual nature. The report does not enable the reader to accept the inference that the gall-bladder was opened. In Mr. *Bryant's* case, there could be no question of the expediency of the operatiou, and the opening into the gall-bladder had presumably already been made through natural processes.

The cases which are valuable as guides for the future are those of *Sims, Tait,* and *Keen.* In all three, the operation was at the outset exploratory, an exact diagnosis not having been made. In two jaundice was present, and these were the cases terminating fatally, in both hemorrhage being an important feature. The absence of evidence of peritonitis in all cases is of great value, as warranting an exploratory incision for diagnostic purposes, where other means have failed, and where an extension of the operation is likely to provide a means for the removal of the cause of the disease.

The absence of any mention of one means of diagnosis in obscure tumors is conspicuous. It would seem of primary importance to use the aspirator in a case of suspected dilatation of the gall-bladder.

*Bartholow** has already aspirated the distended gall-bladder, and the same laws which apply to the use of this instrument in intestinal or vesical puncture may be considered to apply when it is desired to know the contents of a tumor in the region of the gall-bladder.

The great value of the exploratory puncture is to be found in the results of the examination of the fluid thus removed. It seems not unlikely that the expediency of the operation of cholecystotomy in any given case is dependent upon the nature of the contents of the gall-bladder. It is probable that those cases of distended gall-bladder from impacted calculi are most favorable for the operation when the obstruction is at the cystic duct, and where nothing else than a catarrhal fluid is present, as in the case of *Tait.* If the obstruction is in the common duct, or in the hepatic duct, the prognosis becomes more grave, from the obvious difficulty in removing the calculus after the abdomen and gall-bladder have been opened, and from the liability to hemorrhage in consequence of the intense jaundice, which is so sure to follow an obstruction of the common or hepatic duct. There can be no question that this operation is likely to prove of decided benefit in those cases which actually demand it, viz., of distended gall-bladder and cholecystitis, with urgent symptoms from impaction of one or more calculi at the cystic duct. Such are by no means numerous, however; some of them terminate favorably if left alone, and the importance of an accurate diagnosis and a judicious estimate of the serious nature of the symptoms cannot be too urgently insisted upon.

---

* The Cincinnati Clinic, 1877, April 7th, p. 158.

HYPERTROPHIC CIRRHOSIS.

The literature of this subject has been somewhat enlarged during the past year by occasional contributions, among which that by *Brieger*[*] deserves especial mention.

This writer is unable to corroborate the views recently presented so prominently, of the existence of a special form of cirrhosis, whose cause is to be sought for in an irritation proceeding from the bile-ducts. This form of cirrhosis, called hypertrophic, in consequence of the enlargement of the liver, has also been spoken of as insular cirrhosis, because islets of liver, each representing a single lobule, are surrounded by the new-formed connective tissue, which, entering the lobule, causes the destruction of the latter. An abundant network of bile-ducts is to be found within this fibrous tissue.

This hypertrophic cirrhosis is considered to be of biliary origin, and is to be distinguished from the more common form due to the abuse of alcohol. In the latter, the new formation of connective tissue is stated to take place in an annular form, beginning in the vicinity of branches of the portal vein, hence of venous origin. In its growth it surrounds and destroys groups of lobules rather than single ones. The syphilitic interstitial hepatitis is regarded as distinct from the biliary and alcoholic forms, being an admixture of both.

Brieger's observations, as well as the evidence collected by him, show that the abundant new formation of bile-ducts, which has been regarded as characteristic of the biliary, insular, unilobular, or hypertrophic form of cirrhosis, has been found in various affections of the liver. Such new-formed bile-ducts have been seen in acute yellow atrophy, in the atrophic nutmeg liver, and in alcoholic cirrhosis. Although he found this histological condition in several cases of cirrhosis from biliary stagnation, the livers were not hypertrophied, but diminished in size. As the result of his observations, therefore, the new formation of bile-ducts is not characteristic of any one form of cirrhosis, but may occur in the most manifold varieties of chronic interstitial hepatitis.

He was also unable to confirm the statements with regard to the annular distribution of the connective tissue in alcoholic cirrhosis, which has been presented as a means of defining the interstitial hepatitis thus arising.

In examining the early stages of this affection, where death had taken place independently of the hepatic disease, the fibrous tissue was found to occur irregularly in and about the lobules, and also to contain numerous new-formed bile-ducts. The conclusion arrived at by *Brieger* is, that at present it is impossible to classify the different forms of cirrhosis, from their histological appearances.

---

[*] Virchow's Archiv, 1879, Vol. LXXV., p. 103.

These views have been confirmed by *Saundby*,* who states that he has met with cases in which the anatomical appearances described by *Hanot* were present, but the clinical features absent. In one case he had found all the clinical conditions, but the anatomical characteristics were only vaguely defined.

Very recently, Professor *Ackermann*,† of Halle, has maintained that the two forms of cirrhosis, the hypertrophic and the atrophic varieties, are distinct affections in etiology and in anatomical appearances. He sees no distinctive feature in the presence of new-formed bile-ducts, neither does he find the hypertrophic form to be constantly unilobular, nor the atrophic variety multilobular. In the latter affection, the connective tissue may be found within the lobules, as well as between them. He claims that in hypertrophic cirrhosis the development of the fibrous tissue takes place from the normal blood-vessels of the liver, and is not strictly speaking of inflammatory origin, but rather analogous to that observed in elephantiasis. As it does not interrupt the communication between the portal and hepatic veins, and as it does not tend to shrink, this new-formation of fibrous tissue in hypertrophic cirrhosis occasions but little obstruction to the portal circulation.

As the result of his histological study of the atrophic variety of cirrhosis, *Ackermann* claims that the growth of connective tissue proceeds from new-formed, capillary blood-vessels, which arise from the terminal branches of the hepatic artery. He also maintains that a fatty and granular degeneration of liver cells at the edges of the lobule is the irritant which gives rise to this new-formation of connective tissue, which is thus rather of inflammatory origin, and consequently has a tendency to contract. Before this tissue shrinks, there is an enlargement of the liver, and the fibrous tissue surrounds the lobule, and may even enter it for a short distance. The capillaries at the edge of the lobule are early obstructed, to a moderate extent, but with the shrinkage of the new-formed tissue the portal blood becomes eventually completely shut off from that in the hepatic capillaries. The lobular capillaries are then fed only through the hepatic artery, which has thus to serve for the secretion of bile as well as for the nourishment of the liver.

---

* British Medical Journal, Feb. 22d, 1879, p. 285.

† Virchow's Archiv, 1880, LXXX., p. 396.

DISEASES OF THE
EMALE SEXUAL ORGANS.

BY

FRANK P. FOSTER, M.D.,

OF NEW YORK,

CIAN FOR DISEASES OF WOMEN TO THE OUT-PATIENT DEPARTMENT OF THE NEW YORK HOSPITAL.

DISEASES OF THE FEMALE SEXUAL ORGANS.

BIBLIOGRAPHY.

Hegar: Samml. klin. Vortr., No. 105.—Noeggerath: Am. Journ. Obstet., VII., 1875–6.—Croom: Obstet. Journ. Gr. Brit. and Ire., VI., 1878.—Landau: Arch. f. Gynäk., VII., 1874–5.—Gehrung: Am. Journ. Obstet., XI., 1878.—Schultze, B. S.: Centralbl. f. Gynäk., 1878, No. 7.—Sussdorff: Med. Record, XII., 1877.—Atlee: Am. Journ. Med. Sci., Apr., 1871.—Palmer, Vanderveer: Am. Journ. Obstet., VII.—Tait: Lancet, 1879, VII.—Schröder: Centralbl. f. Gynäk., Dec. 20th, 1879.—Fritsch: *Ibid.*, Dec. 6th, 1879.—Coley: Obstet. Journ. Gr. Brit. and Ire., Apr., 1879.—Dawson: Am. Journ. Obstet., VIII.—Winckel: Berichte und Studien, etc., Leipzig, 1874.

Hegar: Die Castration d. Frauen. Samml. klin. Vortr., 1878, Nos. 136, 138.

Norstroem: Hygiea, 1873.—Simon: Berl. kl. Woch., 1875, Nr. 20.—Puech: Ann. de gynéc., May, Aug., 1875.—Leroy: Journ. des connaiss. méd.-chir., 1835, t. II.—Thüngel: Klin. Mittheil., 1860.—Holst: *l. c.*—Jones: Brit. Med. Journ., July 22d, 1865.—Olshausen: Arch. f. Gyn., I.—Breslau: Schweiz. Z. f. Heilk., 2, 1863.—Credé: M. f. Geb., IX.—Jaquet: Berl. klin. Woch., 1874, Nr. 9.—Hegar, *l. c.*—Neudörffer: Zwei neue Fälle von Pyometra und Pyokolpos Lateralis, D. i. Tübingen, 1873.—Atlee: Gen. and Differential Diagnosis of Ovarian Tumors. Philadelphia, 1873.—Nicolaysen: Virchow-Hirsch'scher Jahresb. üb. 1874.—Freund: Zeitschr. f. Geb. u. Gyn., I.—Anderson: Hygiea, 1875.—Veit: Frauenkrankh., zweite Aufl.—Décès: Bull. de la Soc. Anat., July, 1854.—Passauer: Berl. klin. Woch., 1867, Nr. 26.—Neugebauer, *l. c.*—Breisky: Arch. f. Gyn., II.—Braus-Spiegelberg: Berl. klin. Woch., 1874, Nrs. 10 und 11.—Breisky: Arch. f. Gyn., VI.—Magenau: Ein Fall v. Ut. bicornis, etc. D. i. Tübingen, 1872.—Staude: Berl. klin. Woch., 1874, Nr. 22.—Johannovsky: Prager med. Woch., 1877, Nr. 15.—Freund: Zeitschr. f. Geb. u. Gyn., I.—Naecke: Arch. f. Gyn., IX.—Chadwick: Boston Med. and Surg. Journ., Dec. 20th, 1877.—Beronius: Preuss. med. Zeit., 1862, Nr. 33.—Hegar: Monats. f. Geb., XVII.—Hertzfelder: Oesterr. Zeitschr. f. pr. Heilk., Dec. 26th, 1856.—Braun: Wien. med. Woch., 1861.—Teuffel: Ein neuer Fall von Uterus bicornis, etc. D. i. Tübingen, 1874.—Sullivan: Boston Med. and Surg. Journ., Dec. 20th, 1877.—Churchill: Lancet, Nov. 11th, 1865.—Hofmann: Drei Fälle von Uterusmissbildung. Erlangen, 1869, D. i.—Otto: (Quoted by Kussmaul, von dem Mangel, etc.).—Puech: Gaz. des hôp., 1857.—Breisky-Klebs: Arch. f. Gyn., II.—Chátin: Arch. de Tocol., III.—Brown: Boston Med. and Surg. Journ., XCIV.—Emmet: Trans. Am. Gyn. Soc., II.—Young: Obstet. Journ. G. Brit. and Ire., VI.—Emmet: N. Y. Med. Journ., XXIII.—Löhlein: Zeitschr. f. Geb. u. Gyn., I.—Harley: Brit. Med. Journ., 1878, I.—Winsor: Boston Med. and Surg. Journ., XCVI.—Tait: Brit. Med. Journ., 1878, I.—Galabin: Obstet. Journ. Gr. Brit. and Ire., Sept., 1879.

Martin, E.: Zeitschr. f. Geb. u. Frauenkr., I.—Eklund: Nord. med. Arkiv, VIII., 3.—Chrobak: Wien. med. Presse, 1876, 1-12.—Pallen: Am. Journ. Obstet., 1877.—Sims: Trans. Am. Gyn. Soc., III, 1878.—Mundé: Am. Journ. Obstet., VIII.—Kehrer: Arch. f. Gyn., X.—Smith: Obstet. Journ. Gr. Brit. and Ire., V.—Peaslee: Am. Journ. Obstet., IX.—Leblond: Ann. de gyn., May, 1878.—Sims: Trans. Am. Gyn. Soc., III.

Rolly: Ueber die hypertrophische Verlängerung der Vaginalportion des Uterus. D. i. Giessen, 1868.—Dupuy: Arch. de Tocol., Dec., 1875, *et seq.*

Crevet: Beitr. zur Geb., IV., 1875.

Peaslee: Med. Record, XI., 1876.—Bennett: Edinb. Med. Journ., Nov., 1878. —Courty: Arch. gén. de méd., 1876, II.

Guérin: Ann. de gyn., July, 1874.—Olshausen: Arch. f. Gyn., VIII.—Bischoff: Corresp.-Bl. f. Schw. Aerzte, 1878.—Braithwaite: Obstet. Jour. Gr. Brit. and Ire., III.—Braun: Centralbl. f. Gynäk., III., 1879.—Playfair: Obstet. Jour. Gr. Brit. and Ire., Feb., 1879.—Bantock: *Ibid.*, May, 1879.—Atthill: Brit. Med. Journ., Dec. 13th, 1879.—Leopold: Arch. f. Gyn., XII.—Kugelmann: *Ibid.*, X.—Spender: Brit. Med. Jour., 1875, II.—Gallard: Arch. gén. de méd., 1877, II.—Keiller: Edinb. Med. Jour., Oct., 1878.—Mundé: *Ibid.*, March, 1878.

Otterburg: Lettres sur les ulcerations de la matrice, etc. Paris, 1839.—Bennett, J. H.: Pract. Treatise on Inflammation, Ulceration, and Induration of the Neck of the Uterus. London, 1845.—Robert: Des affect. granul., ulcér. et carcin. du col de l'utérus. Thèse, Paris, 1848.—Wagner, E.: Arch. f. phys. Heilk., 1856.—Mayer, C.: Vortr. über Eros., Excor., etc. Berlin, 1861.—Scanzoni: Chronische Metritis. Wien, 1863.—Smith, Heywood: Obstet. Jour. Gr. Brit. and Ire., Dec., 1876.—Wing, C. E.: Brit. Med. Jour., March 16th, 1875.—Halton: Dublin Jour. Med. Sci., June, 1875.—Ruge u. Veit: Centralbl. f. Gyn., 1877, No. 2, and Ztschr. f. Geb. u. Gyn., II.—Hofmeier: Ztschr. f. Geb. u. Gyn., IV., 2.—Atthill: Brit. Med. Jour., 1878, II.

Virchow: V.'s Archiv, 1854, VII.; and Die krankhaften Geschwülste, III., 1, S. 142.—Lebert: Traité d'anat. pathol., I., pl. XL., p. 15.—Cruveilhier: Atlas d'anat. pathol., livr. 39, pl. III., figs. 2 and 2^1.—Wagner: Arch. f. phys. Heilk., 1856, S. 509.—Simon: Mon. f. Geb., XXIII., S. 241.—Höning: Berl. klin. W., 1869, Nr. 6.—Ackermann: Virchow's Arch., XLIII., S. 88.—Martin: Berl. Beitr. z. Geb. u. Gyn., II., S. 51.

Pansch: Reichert's Arch. f. Anat., Phys., etc., 1874, Nr. 6, S. 702.—Frankenhäuser: Corresp.-Bl. f. Schw. Aerzte, 1876, Nr. 14.—Bantock: Lancet, 1878, I., pp. 120 and 162.—Notta: Un. méd., Nov. 14th, 1876.—Williams: Obstet. Journ. of Gr. Brit. and Ire., IV., p. 97.—Campbell: Am. Jour. of Obstet., X., p. 62.—Van de Warker: N. Y. Med. Jour. XXIII., p. 561.—Campbell: Trans. Am. Gyn. Soc., I., p. 198.—Hewitt: Lancet, 1877, II., pp. 871, 907, and 945.—Grenser: Arch. f. Gyn., XI., S. 145.—Studley: Am. Jour. of Obstet., Jan., 1879.—Schultze: Centbl. f. Gyn., III., p. 49.—Foster: Am. Jour. of Obstet., Jan., 1880.

Emmet: Med. Record, Apr. and May, 1871.—Veit, J.: Ztschr. f. Geb. u. Gyn., I., S. 144.—Tauffer: Deutsche med. Woch., 1877, Nr. 22-25.—Bourdon, D.: Des anaplasties périnéo-vagin., etc. Paris, 1875.—Virchow: Verh. d. Berliner Ges. f. Geb., II., S. 205.—Mayer, C.: M. f. Geb., XI., S. 163.—Scanzoni: Beitr. z. Geb. u. Gyn., IV., S. 329.—Braun, C.: Zeitschr. d. Ges. d. Wien. Aerzte, 1864, S. 43.—Martin: M. f. Geb., XX., S. 203.—Barnes: Brit. Med. Journ., Jan. 7th, 1871.—Spiegelberg: Berl. klin. Woch., 1872, Nr. 21 u. 22.—Goodell: Prolapse of the Womb. Phila., 1873; and Some Practical Hints, etc. Med. and Surg. Reporter, Jan. and Feb., 1874.—Lefort: Arch. de Tocol., IV., p. 618.—Braithwaite: Brit. Med. Journ., 1876, II., p. 395.—Galabin: Brit. Med. Journ., 1877, II., p. 408.

Duncan, M.: Edinb. Med. Journ., March, 1877.—Guéniot et Hue: Bull. et Mém. de la Soc. de Chir. de Paris, V., pp. 522 and 530.—Forget: *Ibid.*, p. 559.—Donné: Ann. de Gyn., Jan., 1876.—Duncan: Obstet. Jour. of Gr. Brit. and Ire., V., p. 48.—Tait: *Ibid.*, VI., p. 178.—Watts: Am. Jour. of Obstet., IX., p. 86.—Hennig: Arch. f. Gyn., VIII., S. 214.

Hofmeier: Ztschr. für Geb. u. Gyn., V., 1.—Billroth: Wien. med. Woch., 1876, Nr. 1.—Rein: Arch. f. Gyn., IX., S. 414.—Delore: Gaz. des hôp., Aug. 12th, 1877.—Guéniot: Archiv d. Tocol., II., p. 149.—Henriet: Tribune méd., Dec. 14th, 1879.—Pozzi: Prog. méd., Aug. 14th, 1875.—Meadows: Obstet. Jour. of Gt. Brit. and Ire., III., 561.—Péan: Lancet, July 21st, 1876.—Atthill: Brit. Med. Jour., 1876, II., p. 299.—Kidd: *Ibid.*, 1879, II., p. 240.—Byford: Trans. Am. Gyn. Soc., I., p. 168.—Sims: Trans. Med. Soc. of the State of N. Y., 1875, p. 367.—Emmet: *Ibid.*, p. 207.—Mundé: Am. Jour. of Obstet., IX., p. 157.—Thomas: N. Y. Med. Jour., Mar., 1877.

Sims: N. Y. Med. Jour., Apr., 1874.—Maennel: Prag. Vierteljahrschr., 1874, II., S. 29.—Braun: Wien. med. Woch., 1874, Nr. 39-41.—Hegar u. Kaltenbach: Die operativ. Gyn. Erlangen, 1874, S. 244.—Martin: Verh. d. Breslauer Naturf.-Vers., 1874.—Martin, A.: Ztschr. f. Geb. u. Frauenkr., I., S. 143.—Breisky: Prag. med. Woch., 1877, No. 21.—Emmet: Am. Jour. of Obstet., Jan., 1877.—Thomas: *Ibid.*, Oct., 1877.—Simpson, A. R.: Edinb. Med. Jour., Jan., 1878.—Pozzi: De la valeur de l'hystérotomie. Paris, 1875.

Billroth: Chirurgische Klinik. Berlin, 1879, S. 384.

Olshausen: Arch. f. Gyn., VIII., S. 97.—Schröder: Ztschr. f. Geb. u. Gyn., I., S. 189.—Slavjansky: Obstet. Jour. of Gt. Brit. and Ire., Nov., 1873.—Winckel: Path. d. weibl. Sexualorg. Leipzig, 1878, S. 40, Taf. XX., a.—Bischoff: Corresp.-Bl. f. schw. Aerzte, 1878, Nr. 16.

Müller: Charitéannalen, III. Berlin, 1878, S. 320.—Schröder: Ztschr. für Geb. u. Gyn., III., S. 423.—Credé: Centralbl. f. Chirurg., Aug. 10th, 1878.—von Grünewaldt: Archiv f. Gyn., XI., S. 501.—Leopold: Centralbl. f. Gyn., II., S. 548.—Binswanger: *Ibid.*, III., S. 4.—Gibb: Brit. Med. Jour., 1875, I., p. 204.—Noeggerath: Am. Jour. Obstet., X., p. 105.—Sims: *Ibid.*, July, 1879.

Pichot: Thèse de Paris, 1876.—Brechetti: Gaz. Med. Ital. Prov. Venete, Dec. 27th, 1879; Jan. 3d, 17th, 1880.—Duncan: Med. Times and Gaz., 1879, I., p. 391.

Thomas: Am. Jour. of Obstet., VII., p. 45.—Clay: Lancet, Jan. 6th, 13th, 1877.—Simpson: Obstet. Journ. of Gt. Brit. and Ire., Feb., 1876.

Scanzer: Ztschr. f. Geb. u. Gyn., IV., S. 313.—Beigel: Krankh. d. weibl. Geschl., S. 504.

Fasbender: Ztschr. f. Geb. u. Frauenkr., I., p. 125.—Drysdale: Obstet. Jour. of Gt. Brit. and Ire., II., p. 422.—Tilt: Brit. Med. Jour., II., p. 293.—Duncan: Med. Times and Gaz., 1879, I., p. 221.—Graham: Boston Med. and Surg. Jour., XCIV., p. 146.—Abbot: *Ibid.*, Nov. 14th, 1878.—Piffard: Med. Record, XIII., p. 204.

Finkel: Virchow's Arch., LXIII., S. 401.—Mayer, L.: Berl. Beitr. z. Geb. u. Gyn., IV., S. 28.—Hoggans: Arch. f. Gyn., X., S. 301.—Beigel: *Ibid.*, IX., S. 84.—Hennig: *Ibid.*, VIII., S. 342.—Leopold: *Ibid.*, X., S. 293.—Haussmann: *Ibid*: IX., S. 154.—Rothe: Memorabilien, XXIV., 11.—De Sinéty: Archiv d. Tocol., III., p. 510.—Williams: Obstet. Jour. of Gt. Brit. and Ire., V., p. 251.

Hausammann: Ueber Retentionsgeschwülste schleim. Inh. in d. weiblichen Genitalien. Zürich, 1876.

Wibaille: Des hernies de l'ovaire. Thèse de Paris, 1874.—Puech: Ann. de Gyn., Nov., 1878.—Balleray: Am. Jour. of Med. Sci., Oct., 1877.

Gallez: Histoire de kystes de l'ovaire, etc. Bruxelles, 1873.—Kœberlé: Mal. des ovaires, etc. Extrait du Nouveau Dict. de Méd. et de Chir. Prat. Paris, 1878, t. XXV.—Olshausen: Krankh. d. Eierstöcke. Stuttgart, 1877.—Friedländer: Beitr. z. Anat. d. Cystovarien. D. i., Strassburg, 1876.—Marchand: Beitr. z. Kenntn. d. Ovarientumoren. Halle, 1879.—Malassez et de Sinéty: Arch. de Physiol., 1878, Nos. 1 et 4.—Leopold: Arch. f. Gyn., VI., S. 232.—Kœberlé: Gaz. méd. de Strasbourg, 1874.—Röhrig: Dtsch. Arch. f. klin. Med., XVII., S. 340.—Thornton: Med. Times and Gaz., July 28th, 1877.—Veit: Tageblatt d. Casseler Naturf. Vers., 1878, S. 167.—Spiegelberg: Arch. f. Gyn., I., S.

146.—Schatz: *Ibid.*, IX., S. 115.—Geissel: Dtsch. med. Woch., 1877, Nr. 43.—Heer: Ueber Fibrocysten des Uterus. Zürich, 1874.—Stahl: Centralbl. f. Gyn., III., S. 78.—Schultze: *Ibid.*, S. 129, Jan. 3d, 1880.—Stahl: *Ibid.*, I., p. 145.—Kœberlé: Bordeaux méd., Jan. 11th, 1876.—Tait: Lancet, Feb. 7th, 1880.—Noeggerath: Am. Jour. of Obstet., Jan., 1880.

Stimson: Am. Jour. of Med. Sci., Jul, 1879.—Mundé: Trans. Am. Gyn. Soc., II., p. 348.—Semeleder: N. Y. Med. Jour., XXIII., p. 572.

Hegar: Samml. klin. Vortr., Nr. 109.—Olshausen: *Ibid.*—Bocker: Ztschr. f. Geb., I., S. 353.—Kaltenbach: *Ibid.*, S. 537.—Baum: Centralbl. f. Gyn., II., S. 73.—Pauli: Berl. klin. Woch., Nov. 23d, 1874.—Stilling: Dtsch. med. Woch., 1877, Nr. 10, 11, 12, 13.—Ahlfeld: *Ibid.*, Jan. 3d, 10th, 17th, 1880.—Duplay: Arch. gén. de méd., 1878, II., p. 742.—Labbé: Bull. et Mém. de la Soc. de Chir. de Paris, V., p. 767.—Tait: Obstet. Jour. of Gr. Brit. and Ire., Apr., 1879.—Smith: *Ibid.*, Nov., 1879.—Keith: Brit. Med. Jour., 1878, I., p. 8; II., p. 590.—Thornton: *Ibid.*, 1878, I., p. 125.—Wells: *Ibid.*, 1877, II., p. 837.—Bantock: *Ibid.*, 1877, II., p. 436.—Thornton: Lancet, 1879, I., pp. 307, 418, 460.—Keith: *Ibid.*, 1876, I., p. 562.—Wells: *Ibid.*, 1877, I., p. 315.—Doran: St. Barthol. Hosp. Rep., XIV.—Kimball: Trans. Am. Gyn. Soc., II., p. 525.—Byford: Am. Journ. of Obstet., Jan., 1879.—Atlee: Am. Jour. of Med. Sci., Jan., 1877.—Wing: Boston Med. and Surg. Jour., XCV., p. 516.—Fitch: N. Y. Med. Jour., Apr., 1875.—Lee: *Ibid.*, XXIII., p. 255.—Sims: Med. Record, XI., p. 792.

Pauly: Berl. Beitr. z. Geb. u. Gyn., IV., S. 3.—Cousin: Thèse de Paris, 1877.—Byford: Trans. Am. Gyn. Soc., III., p. 162.

Atlee: Trans. Am. Gyn. Soc., II., p. 326.

Birch-Hirschfeld: Lehrb. d. path. Anat., S. 1101.—Marchand: Beitr. z. Kenntn. d. Ovarientumoren. Halle, 1879, fig. 4.

Eidam: Arch. f. klin. Med., 1875, 3, 4.—Schlesinger: Oesterr. med. Jahrb., 1878, 1, 2.—Besnier, J.: Arch. gén. de méd., 1878, II., p. 257.—Guérin, A.: Progr. Méd., 1879, No. 41.—Duncan: Med. Times and Gaz., 1879, I., pp. 112, 167.—Van de Warker: Trans. Am. Gyn. Soc., III., p. 337.—Budd: Am. Jour. of Obstet., IX., p. 94.—Brickell: Am. Jour. of Med. Sci., Apr., 1877.—Octerlony: Va. Med. Month., IV., p. 516.

Martineau: Tribune méd., Dec. 28th, 1879.—Macquibban and Ogston: Brit. Med. Jour., 1879, I., p. 543.—Minot: Boston Med. and Surg. Jour., May 25th, 1876.—Goodell: Med. Record, XIII., p. 67.—Harrison: Va. Med. Month., Oct., 1875.

Schatz: Arch. f. Gyn., IX., S. 120.—Gusserow: *Ibid.*, S. 478, u. X., S. 184.—Arning: *Ibid.*, X., S. 392.

Wiener: Arch. f. Gyn., XI., S. 572.—Haussmann: *Ibid.*, XII., S. 163.—Villard: Ann. de Gyn., IX., p. 118.———: Arch. gén. de méd., 1876, I., p. 481.

Ruge, C.: Ztschr. f. Geb. u. Gyn., IV., S. 133.

Schippert: A Treat. on Vesico-vag. Fist. New Orleans, 1866.—Simon: Wien. med. Woch., 1876, Nr. 27-32.—Bozeman: Am. Jour. of Med. Sci., July, 1870 Ann. de Gyn., Aug., 1876; Obstet. Jour. of Gr. Brit. and Ire., June-Aug., 1877.—Bandl: Wien. med. Woch., 1875, Nr. 49-52, and 1877, Nr. 30-32.—Hempel: Arch. f. Gyn., X., S. 479.—Amabile: Un Osserv. di Fistola ves.-vag., etc. Napoli, 1872; Ann. de Gand, 1876, No. 13.—Bouqué: Du Traitement des Fistules Uro-Génit., etc. Paris, Gand, et Leipzig, 1875.—Martin: Ztschr. f. Geb. u. Gyn., IV., S. 320.—Herrgott: Arch. de Tocol., II., p. 336.—Simon: Obstet. Jour. of Gr. Brit. and Ire., IV., pp. 435, 497, 589.—Maunder: Lancet, 1876, I., p. 883.—Bozeman: *Ibid.*, 1876, II., p. 633.—Campbell: Trans. Am. Gyn. Soc., p. 354.

Lutard: Du Vaginisme, etc. Paris, 1874.—Fritsch: Arch. f. Gyn., X., S. 547.—Duncan: Med. Times and Gaz., 1878, II., p. 453.—Thomas: Hosp. Gaz., II., p. 227.

Herrgott: De l'exstrophie vésicale dans le sexe féminin. Paris, 1874.—Ogston: Med. Times and Gazette, 1876, I., pp. 161, 189.—Tait: Trans. Am. Gyn. Soc., I., p. 318.

Huguier: Mém. de l'Acad. de Méd., 1849, T. XIV.—Bernutz: Arch. de Tocol., July, 1874.—Siredey: Un. méd., Oct. 3d, 12th, 1876.

Jüterbogk: Arch. f. klin. Chir., XXIV., S. 108.—Liebmann: Ztschr. f. Geb. u. Gyn., I., S. 393.—Hue: Arch. de Tocol., III., pp. 341, 590.—Bantock: Obstet. Jour. of Gr. Brit. and Ire., IV., p. 655; V., p. 94.—Chambers: *Ibid.*, V., p. 34.—Maunder: Med. Times and Gaz., 1875, II., p. 40.—Reamy: Trans. Am. Gyn. Soc., II., p. 576.—Jenks: Am. Jour. of Obstet., April, 1879.—Brickell: Am. Suppl. to Obstet. Jour. of Gr. Brit. and Ire., II., p. 145; Am. Jour. of Med. Sci., Apr., 1875.

Gellé: Tribune méd., Dec., 1875.—Thomas: Hosp. Gaz., III., p. 62.—Gill: St. Louis Med. and Surg. Jour., Dec., 1875.

GYNÆCOLOGICAL EXAMINATION.

Bilateral incision of the cervix is often the readiest and least dangerous way of reaching the uterine cavity. Tupelo is included among the dilating agents that have come into use recently.

Attempts to inspect the mucous membrane of the cervical canal usually fail, unless there is laceration with eversion, or, unless this condition is brought about artificially by bilateral incision. In case the cervix is enormously dilated, the canal may be explored by means of Simon's urethral specula.

In many cases of abdominal tumors, particularly where there is free fluid in the abdominal cavity, or where there is a flaccid cyst (after puncture), percussion is of great service, as well as in cases where there are obstacles to satisfactory palpation. Auscultation is often of much avail in distinguishing large tumors from the gravid uterus. The so-called uterine souffle is not infrequently heard in cases of large fibroids, but only exceptionally with ovarian tumors. Signs of friction, most frequent in ovarian cysts, as the result of roughening of the peritoneal surfaces from recent inflammation or papillary outgrowths, are better felt than heard. A metallic succussion sound is heard in connection with intestinal perforation and peritonitic exudation, and when gas has formed in ovarian cysts.

HÆMATOMETRA.

For evacuating the uterus in cases of hæmatometra, *Simon* and *Spiegelberg* advise puncture through the bladder, beneath the anterior fold of the peritonæum, the urethra having first been dilated. Whether previous puncture of the tube-sac, made adherent to the abdominal wall, as practised by *Haussmann* (*Zeitschr. f. Geb. u. Gyn.*, II.), enables us to avoid dangerous rupture of the sac is more than doubtful.

STENOSIS OF THE UTERUS.

The cervical mucus, if very thick, may not flow readily through a very narrow os uteri, but may collect above it and so distend the cervical canal that, after incising the os, the finger may be introduced, even in nulliparæ. Chronic inflammation not uncommonly results from the irritation of the uterus occasioned by severe dysmenorrhœa, and slight contractions then cause severe pain.

The ordinary operations open only the vaginal portion of the cervix. However, the os externum is the most common seat of the constriction. When the latter extends up to the os internum, we should first secure sufficient patency of the external os, and then proceed higher. For this purpose *Schröder* uses *Martin's* double-bladed hysterotome. The tendency to re-contraction is very great. He combats it by the repeated introduction of large copper sounds, but it is difficult to secure permanent dilatation. The insertion of a sponge-tent directly after the incision is too dangerous to be recommended. *Courty** suggests bringing the investing and the lining mucous membranes together with sutures, but *Schröder* has found it answer better to sew the cut surfaces directly together.

Simple lateral incision does not always answer the purpose, and we have to resort to rather complicated operations. If the cervix is much too long, its tip should be cut off.

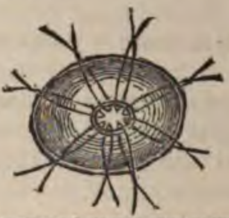


FIG. 1.—Peripheral suture after amputation.

For *amputation of the vaginal portion of the cervix*, the organ should be drawn down with hooked forceps until it appears at or just within the vaginal orifice. If there are no adhesions, this manœuvre, carefully practised, is wholly devoid of danger. The cervix is then cut through, at the point decided upon, by a free use of the knife. The investing membrane is then stitched to the lining membrane, so as to cover the stump (Fig. 1). If the parenchyma is very thick, and the cervical canal narrow, the lining membrane of the anterior half is to be stitched to the corresponding portion of the investing membrane, and the like procedure is to be carried out upon the posterior lip. The remaining loose edges of the investing membrane are then to be stitched to each other at either side. This method, taught by *Hegar*,* *Simon*, and *Spiegelberg*,† gives a good stump, and secures certain and permanent cessation of hæmorrhage (Fig. 2). Even if the cervical canal is dilated above the os externum, it is best to adopt this method, as it secures such gaping of the canal as to prevent stagnation of the secretion.

---

* Gaz. hebdom., May 9th, 1873.

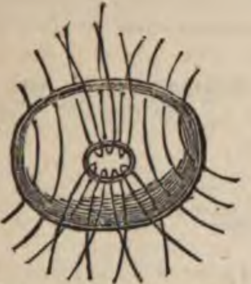


FIG. 2.—Suture after wedge-shaped excision of the two lips.

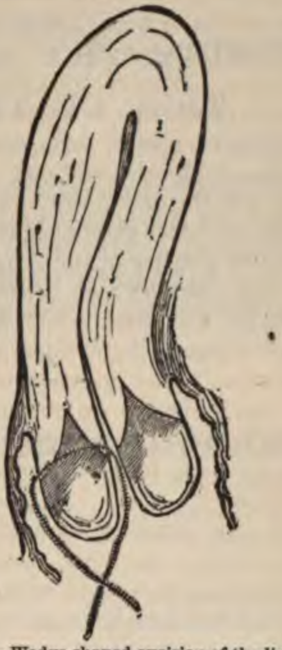


FIG. 3.—Wedge-shaped excision of the lips.

If the tissue of the cervix is very thick, as it often is where there has been inflammation, we must make its very wall thinner, to take off the pressure from the cervical canal. To do this, we proceed in the manner somewhat elaborately described by *Marckwald* ‡ as *Simon's chine-shaped* [kegelmantelförmige] *excision*. The same purpose may be answered very simply by first slitting up the cervix on either side, and then removing a wedge-shaped portion from each lip—the point of the wedge looking upward and situated about one cm. higher than the incision through the mucous membrane. The flaps are then stitched together, and the remains of the bilateral cleft are also closed with sutures § (Fig. 3).

---

* Monatsschr. f. Geb., XXXIV., p. 395; Tagebl. d. Wiesbadener Naturforscherv., 1873, p. 176; Die operative Gynækologie. Erlangen, 1874, p. 237; and ODEBRECHT: Berl. Beitr. z. Geb. u. Gyn., III., p. 220.

† Archiv f. Gyn., V., p. 440.

‡ Archiv f. Gyn., VIII., p. 48.

§ *Schröder* has operated in this way for several years past. *Howitz* (Centralbl. f. Gyn., 1878, No. 11) employs a quite similar proceeding.

Kehrer[*] makes from six to eight radiating incisions into the cervix.

Amputation with the knife has several advantages over that with the galvanic-cautery loop, as recommended by *Spiegelberg.*[†] Patency of the os is more likely to be maintained, and bleeding is better controlled with sutures than with the cautery.[‡]

HYPERTROPHY OF THE VAGINAL PORTION OF THE CERVIX UTERI.

To prevent hemorrhage in amputation, the galvanic-cautery loop may be used, but it is better to operate with the knife, using as a tourniquet a cord tied around the cervix above two needles passed through its substance at right angles to each other. For this purpose it is best to use an elastic cord, as recommended by *A. Martin.*[§]

CHRONIC METRITIS, UTERINE INFARCTION.

TREATMENT.

Deep puncture of the cervix, as recommended by *Spiegelberg,*[‖] seems to offer no advantage over scarification. In using iodine, it is best to paint the upper portion of the vagina with equal parts of the tincture (Ph. Bor.) and glycerine, as recommended by *Breisky.*

ENDOMETRITIS.

DIAGNOSIS.

Vegetations should be sought for by dilating the os internum or incising the os externum, and then exploring the cavity with the finger, when the mucous membrane will be found thickened, softened, and smooth or covered with tuberose inequalities. It is simpler to pass a very small curette through the undilated cervix, scrape away some of the mucous membrane, and examine it with the microscope.

---

[*] Archiv f. Gyn., X., p. 431.

[†] *Ibid.*, V., p. 436; also BYRNE, J.: Trans. Am. Gyn. Soc., 1878, p. 57, and LEBLOND: Traité élém. de chir. gyn. Paris, 1878, p. 468.

[‡] *Moericke, S.:* Zeitschr. f. Geb. u. Gyn., III., p. 328.

[§] Berl. klin. Woch., 1876, Nr. 4.

[‖] Arch. f. Gyn., VI., p. 484.

ENDOMETRITIS CERVICIS.

ETIOLOGY.

Childbed is a fertile source of cervical disease. While not prepared, says *Schröder*, to join with those who, since *Emmet's* observations, assume that ectropium following laceration keeps up an irritated state of the everted mucous membrane and disposes it to disease, but rather believing that the membrane, no longer irritated, becomes hardened and covered with pavement epithelium; yet, no doubt, the cicatricial contraction of the lacerations affects the vascular supply of the mucous membrane unfavorably, so that certain parts remain swollen and irritated.

SYMPTOMS.

The relation of *laceration of the cervix* to ectropium* and to chronic catarrh of the mucous membrane is still an open question, says *Schröder*. He cannot quite admit the simple explanation that the exposure of the mucous membrane consequent on the laceration is the direct cause of the inflammation. He would say rather, severe lacerations do not necessarily cause ectropium, but they favor its occurrence, which becomes specially marked in cases of malposture of the uterus. The eversion of the mucous membrane is most marked when there is concomitant cervical catarrh; of itself, however, laceration never gives rise to catarrh, but, when the healthy mucous membrane is thus exposed to irritation, its cylindrical epithelium is converted into flat epithelium, as may be seen in the case of a procident uterus. For the superficial and deep extension of the cylindrical epithelium, the peculiar irritation of catarrh is necessary; however, with the gaping of the lips due to lateral lacerations, the proliferative tendency of the catarrhal mucous membrane is specially pronounced, but, as we should never forget, also specially noticeable.

Cervical catarrh of long standing is apt to cause sterility, but this does not always occur, at least in women who have already borne children. It is said to be due to the profuse secretion blocking the progress of the semen, and to the hyperplastic formations that choke the cervical canal.

In marked cases in nulliparæ, it is almost always present, due, no doubt, in great degree to the thick plug of mucus that fills the dilated cavity of the cervix. The disease is eminently chronic. Spontaneous recovery, which is rare, is not likely to occur until after the menopause, yet at that period of life the most pronounced cases are very common. When the disease subsides, the mucous membrane becomes atrophic, and only little nodules remain in place of the swollen follicles. Even the calcareous deposits disappear.

---

* ROSER: Arch. d. Heilk., 1861, S. 97.—SMITH, TYLER: Pathology and Treatment of Leucorrhœa. London, 1855, p. 84.—EMMET: Surgery of the Cervix, etc. New York, 1862; Laceration of the Cervix Uteri. Am. Jour. of Obstet., Nov., 1874.—BREISKY:—Wien. med. Woch., 1876, Nr. 49–51.

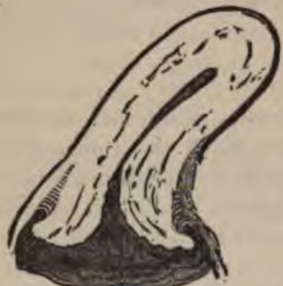


Fig. 4.—Marked ectropium, with catarrh.

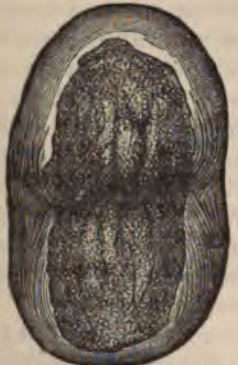


Fig. 5.—The same seen through the speculum.

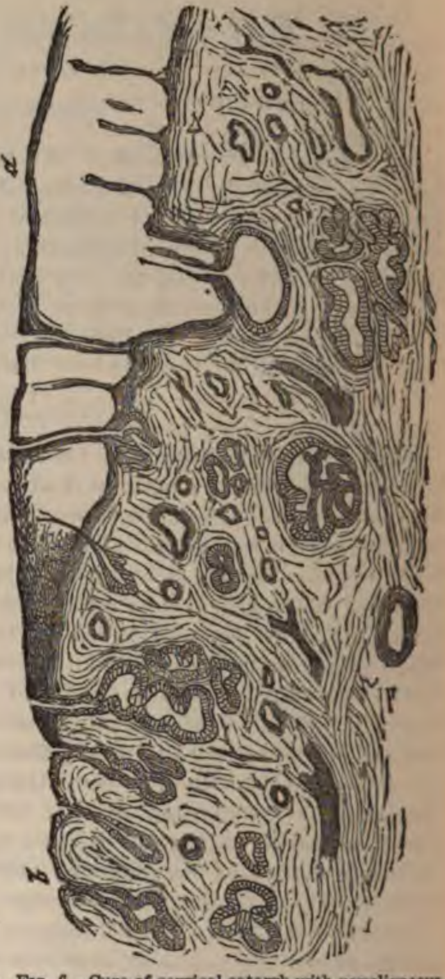


Fig. 6.—Cure of cervical catarrh with pyroligneous acid.

DIAGNOSIS.

Ocular examination gives the most trustworthy data. Little is gained, however, by the use of the uterine specula. It is better to introduce Sims' speculum, pull the uterus down with forceps, and force the lips apart. Bilateral incision is strongly to be recommended, as it is the first step in efficient treatment. It is hard to say whether the glandular neoplasm is still benign or the beginning of carcinoma. Digital examination is here of little service. If, however, we see through the speculum gray, white, or yellowish follicles, and the mucous membrane is free from ulceration, we may exclude advanced carcinoma.

TREATMENT.*

In cases that are not too far advanced, a cure may follow after several weeks of treatment. We should first do away with all sources of irritation—proscribe severe exertion, regulate the bowels and forbid cohabitation. Repeated punctures of the affected mucous membrane are very important, particularly of the swollen follicles, but, as a rule, they are not enough. Pyroligneous acid is the best application for eroded surfaces, used daily for several weeks. Figure 6 shows the improvement produced by a month of such treatment. Whilst at *a* (the site of the former erosion) thick pavement epithelium is already to be seen, at *b* (toward the interior of the cervix) the catarrhal changes are still marked.

To restore the cervical mucous membrane to its normal condition is much more difficult. In slight cases this may be done with irritants, but their application to the whole diseased cervix is by no means easy, owing to the narrowness of the os externum. Where the latter is very large, or where there is eversion, the medicaments may be poured in through a cylindrical speculum; otherwise the cavity of the cervix must be made accessible. Sometimes it will suffice to draw the lips apart with forceps or hooks. If not, intra-cervical injections or the use of the applicator may be resorted to. The latter may be used to introduce the liquid into the cervix after pouring it into the speculum. The irritants employed are, pyroligneous acid, acid nitrate of mercury, solutions of nitrate of silver, tincture of iodine, chloride of iron, and nitric acid. These agents suffice to cure in light cases, but the severer ones, with marked glandular new-formation and adenoma-like outgrowths, resist them all. Even with fuming nitric acid and the free use of the cautery-iron, their radical cure is very difficult. The repeated use of sponge-tents, too, does not wholly destroy the growths, much less prevent their recurrence. In such cases, excision of the diseased mucous membrane cannot be praised too highly. In cases of long standing, it is often doubly indicated when the inflammatory irritation has led to irregular growths of the cervical parenchyma. The uterus is to be drawn down, and the cervix divided bilaterally up to the vaginal insertion. The lips should now be drawn apart so as to allow of inspection of the whole canal as far as the os internum. A transverse incision is then made through the mucous membrane of either lip, as high up as possible (see Fig. 7 *a*), and these incisions should extend into the parenchyma. The mucous membrane and some of the underlying tissue are then cut away, from the tips of the flaps, *c*, to the first incisions. What remains of each lip is then turned in upon itself, and so secured by sutures. The resulting cervical cavity is thus almost wholly covered with vaginal epithelium, which has no tendency to become diseased. If the lips are very much enlarged, the superfluous portions may be removed at the same time.

---

* These remarks, as well as all expressions of opinion given in the gynæcological section of this work, are to be interpreted as the views of SCHRÖDER, unless reference is made to some other author.—F. P. F.

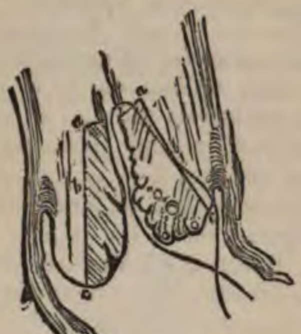


FIG. 7.

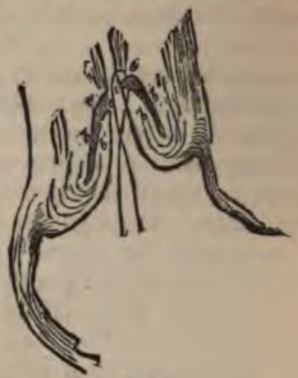


FIG. 8.

FIG. 7.—Line of incision in removal of the cervical mucous membrane.
FIG. 8.—Application of the sutures.

In cases of deep laceration, the operation known as Emmet's may be combined with this procedure. This consists in refreshing the edges of the laceration and uniting them with sutures. A strip of mucous membrane should be left at the middle, wide enough to leave the cervical canal of sufficient size and somewhat the larger at the os externum. Complete union generally follows, but it is of no consequence if little fistulous openings are left.

Irrespective of cervical catarrh, a peculiar pathological importance attaches to extensive lacerations, for, when the abdominal pressure is high, there are symptoms of tension at the angles of the rent—either local pains, incapacity for standing and walking, or nervous derangements of various kinds. Emmet's operation is often indicated for these reasons alone, but it does not cure the catarrh that may also be present, for even in Emmet's own practice the latter requires treatment after the operation. It is in just these cases that a permanent cure of both the catarrh and the laceration may be attained by combining Emmet's operation with excision of the mucous membrane.

CHANGES IN THE ATTITUDE AND SHAPE OF THE UTERUS.

The question first arises: What is the normal attitude of the uterus? This is not so easily answered. Post-mortem changes, even in those recently dead, interfere materially with the results of investigations upon the cadaver, owing to the softening of the organ that follows, changes of the intra-abdominal pressure, and the like. We are therefore compelled to resort to examinations of living subjects, and here, except in the rare instances of laparotomy with a normal state of the genitals, we are confined to the sense of touch. Bimanual palpation, however, answers all practical purposes under favorable circumstances.* It shows that, with the bladder empty and the rectum either empty or but moderately filled, the uterus is turned decidedly forward, so that its fundus rests behind the upper part of the symphysis pubis (Fig. 9), and that this, its ordinary posture, is subject to very great physiological variations. The same is true of its position, as it rises and falls with changes in the intra-abdominal pressure, even with the movements of respiration.

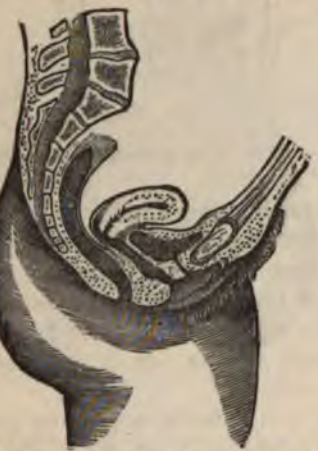


FIG. 9.—Normal attitude and shape of the uterus.

When this pressure gives place to suction, the uterus may rise high into the abdomen, the vagina becoming filled with air. The attitude of the uterus is specially affected by different states of the bladder, as to repletion or the reverse, and to a less degree by those of the rectum. The coils of small intestine in Douglas's space form the filling-in material, rising when the bladder and rectum are filled, and falling when they are empty.† As the bladder fills, the uterus becomes more erect and is pressed somewhat backward. When the bladder is highly distended (Fig. 10), the posterior surface of the uterus is pressed against the rectum, and the intestines leave Douglas's space. When the rectum is distended, the cervix is pushed forward somewhat, the body of the organ being but little influenced. The connections of the uterus with neighboring organs are so yielding that the cervix is readily moved forward and the fundus backward to a considerable degree.

---

* *B. Schultze* (Centralbl. f. Gyn., 1878, Nr. 11) has described a method of examination by which the attitude of the uterus may be made out with great precision in the living subject.

† It is thought by many observers that Douglas's pouch seldom contains small intestines, and such is the opinion of the present writer.

Accordingly, marked changes in the attitude of the uterus must be looked upon as physiological, provided they are due to recognizable, transitory conditions, upon the subsidence of which the organ resumes its wonted posture. Pathological changes of this sort are characterized by their persistence or by some abnormality in the shape of the uterus. Normally, it is somewhat anteflexed, the degree of flexion varying but very little under normal conditions. If, with the body of the organ in its proper relations, the flexion is diminished, we have *anteversion;* if it is increased, *anteflexion.* A persistent inclination of the corpus uteri backward is always pathological. If, with this backward displacement of the fundus, the normal anteflexion is preserved or simply straightened out, we have *retroversion;* if the organ is bent backward upon itself, *retroflexion.*

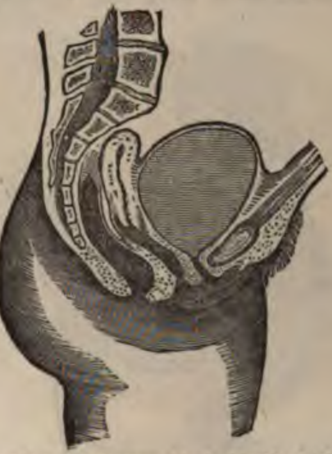


FIG. 10.—Attitude of the uterus with the bladder highly distended.

PROLAPSE OF THE VAGINA, PROLAPSE OF THE UTERUS, HYPERTROPHY OF THE CERVIX.

TREATMENT.

Anterior colporrhaphy (see Fig. 11) consists in cutting out an oval piece from the mucous membrane of the anterior vaginal wall and uniting the borders of the wound by suture. The piece should not be too small, but should reach from the urethral prominence to just in front of the anterior lip of the cervix. To avoid wounding the bladder, and to guard against severe hemorrhage, the tissue should not be too deeply incised.

Schröder uses a knife of pretty good size, double-edged, and pointed like a lancet, which he passes under a broad strip of the mucous membrane, and then cuts laterally. Others prefer to dissect the flap up, which is apt to leave rather an uneven surface, or to tear it away from the subjacent tissue, whereby severe hemorrhage is most easily avoided. After levelling down the surface carefully with scissors, the sutures are applied —both superficial and deep, the former (Fig. 11, *b*) securing accurate coaptation of the cut edges of mucous membrane, and the latter (Fig. 11, *a*) preventing the formation of a pocket in which blood and discharges might collect. Not more than three deep sutures are needed generally. The superficial sutures must be numerous, however, as primary union throughout is of the highest importance. The superficial sutures should be tied first, and never should one of the deep ones be tied until the two adjacent superficial stitches have been tied.

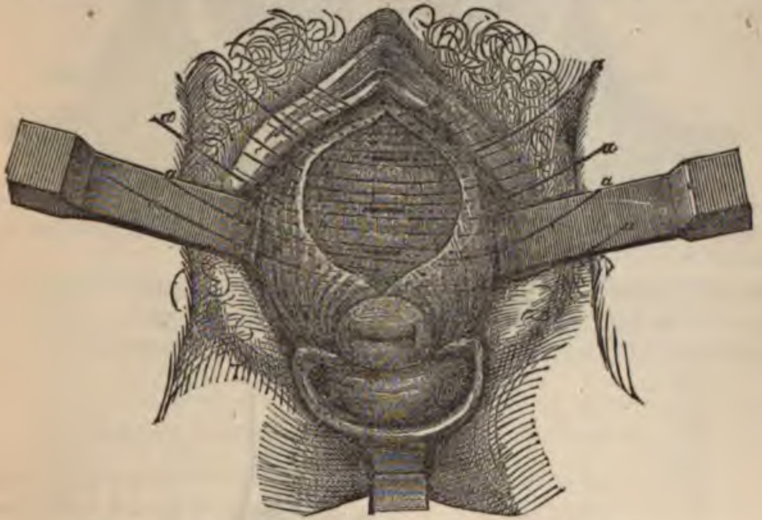


FIG. 11.—Anterior Colporrhaphy. *a*, deep; *b*, superficial sutures.

Posterior colporrhaphy consists in the removal of a part of the posterior vaginal wall and the whole posterior portion of the vaginal entrance. As shown in Fig. 12, the denudation should extend to within a few centimetres of the very summit of the vaginal vault. At the perineum, the incisions should curve backward to a point, otherwise a pouch-like projection would be formed on bringing the wound together. The surface to be denuded is large, and the process tedious, so that considerable loss of blood must be expected. The sutures are applied quite as in anterior colporrhaphy, a few deep and some moderately deep ones being inserted between the superficial ones. The operation produces such a forward curvature of the posterior wall of the vagina that, in the standing posture, it runs almost horizontally forward to the markedly narrow introitus, and the anterior vaginal wall and the uterus rest upon it (Fig. 13).

To save time and to heighten the effect, Schröder combines amputation of the cervix with anterior colporrhaphy in a somewhat modified procedure :* he first splits the cervix bilaterally, then performs wedge-shape amputation of the posterior lip, and finally anterior colporrhaphy in such a manner that the anterior lip falls within the denuded region, and the upper end of the oval, therefore, comes to lie in the cervical canal. The denudation must be made much deeper at the situation of the anterior lip,

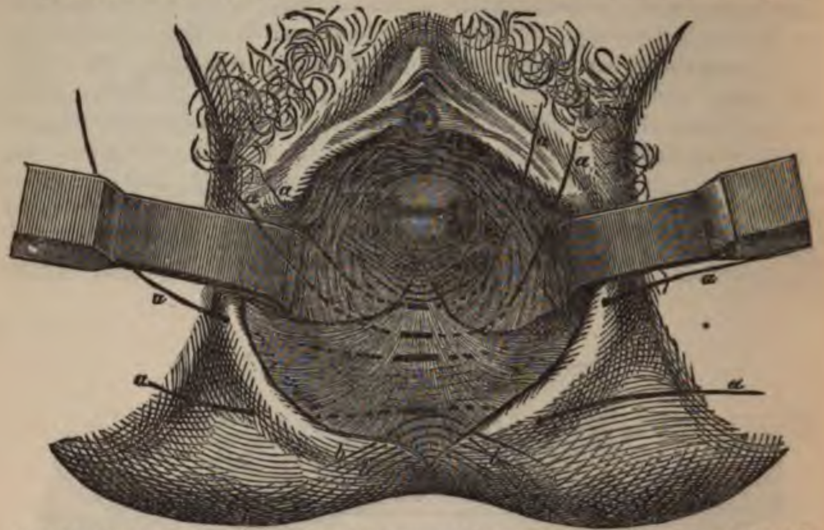


FIG. 12.—Posterior Colporrhaphy. *a*, deep sutures ; *b*, superficial sutures, shown only at the lowermost portion of the perineum.

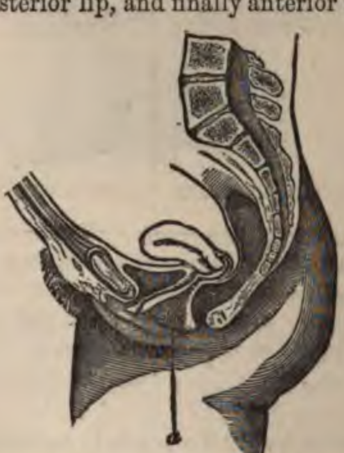


FIG. 13.—Result of posterior colporrhaphy. *a*, the newly-formed floor of the pelvis.

---

* See Ztschr. f. Geb. u. Gyn., III., S. 424.

for, if the lip were left, the tension would be too great. The advantage is, that the posterior wall of the cervix rests upon the upper part of the vaginal cicatrix.

INVERSION OF THE UTERUS.

DIAGNOSIS.

In inversions due to neoplasms, there is danger of overlooking the fact that the stalk of the tumor is formed by the inverted uterus. The uterus is of a deeper color and softer than the tumor, and bimanual examination will obviate error.

MYOMATA (FIBROMYOMATA, LEIOMYOMATA, FIBROIDS) OF THE UTERUS.

ETIOLOGY.

As regards age, the following table embodies *Schröder's* observations for the three years preceding the publication of his last edition :

AGE.	PRIVATE PRACTICE.	POLYCLINIC.	TOTAL.
20–30	15	15	30
30–40	62	35	97
40–50	104	51	155
50–60	15	27	42
60–70	—	2	2
over 70	—	1	1
Total	196	131	327

Winckel's attempt* to account for the occurrence of myomata by attributing them to irritation, local or general, has not proved successful. When we reflect that these causes are in daily operation, their connection with tumors becomes very doubtful.

TREATMENT.

Of all internal remedies, ergot alone is to be recommended, as revived by Hildebrandt.† It should be used energetically. It is best to inject under the skin of the abdomen, every other day, as much as a Pravaz's syringe will hold of a one-to-five solution of doubly purified ergotin, which keeps better if a drop of carbolic acid is added. Generally the injections have to be kept up for months together, scarcely any effect being seen until fifty have been given, and occasionally none until hundreds have been administered. The process is tedious and painful. Although a good, fresh preparation may not cause abscesses, chronic indurations and painful lumps are very common, and the treatment often has to be discontinued, in sensitive women, from the great pain or from fever due to the many inflamed spots. The more superficial are the injections, the more likely are these results to follow. The cases must be selected. Soft tumors are most readily affected by uterine contraction, while old, hard, subserous myomata afford no hope for benefit from ergotin. Although few wholly deny the effect of this treatment, it is not to be depended upon. The hæmostatic action is occasionally quite striking. It is not rare for the growth of a tumor to be arrested; far less frequently does one grow smaller; and the entire disappearance of a tumor is recorded in but very few questionable cases. *Schröder* has never seen it. From the great danger of operative interference, as a rule, from the far greater hopelessness of other remedies, and from the gravity of the affection, he thinks, nevertheless, that this treatment should be thoroughly tried in suitable cases. The officinal preparation very often causes much inflammation and even suppuration. To avoid this, *Wernich*[*] has recommended a preparation, made by freeing powdered ergot of oil and of matters soluble in alcohol, extracting it with water, and then removing mucous and other non-essential material by diffusion through parchment-paper. We cannot yet say whether Dragendorff's sclerotinic acid (0.04 in five times its weight of water once or twice a day) has any advantages; certainly its employment is not painless. Many other drugs have been recommended, but their action is very problematical. Those most used are iodine, iodide of potassium, bromide of potassium, and chloride of calcium (especially by the English, who think it favors atheromatous degeneration of the vessels). *Guéniot*[†] recommends arsenic and phosphorus to induce fatty degeneration. Many look for a diminution or even disappearance of the tumors from the use of mud baths, especially those containing iodine and bromine. Others employ electricity.

---

* Samml. kl. Vortr., Nr. 98. See also RÖHRIG: Berl. klin. Woch., 1877, Nr. 30 u. 31, and ENGELMANN: Ztschr. f. Geb. u. Gyn., I., S. 130.

† Berl. klin. Woch., 1872, Nr. 25; Berl. B. z. Geb. u. Gyn., III., S. 263; Am. Jour. of Obstet., VII., p. 529.—BENGELSDORF: Berl. klin. Woch., 1874, Nr. 2.—KEATING and ASHHURST: Am. Jour. of Med. Sci., July, 1873.—GOODELL: Report

on the Progress of Obstet. and Gyn., 1873, p. 24.—Chrobak: Arch. f. Gyn., VII., S. 293.—Fehling: L. c., S. 384.—Dean: Am. Jour. of Obstet., VII., p. 265, and Proc. of Phila. Obstet. Soc.—Winckel: Klin. Vortr., Nr. 98.—Leopold: Arch. f. Gyn., VIII., S. 182.—Geissel: Dtsch. med. Woch., 1877, Nr. 44.—Byford: Address in Obstet., etc. Phila., 1875.—Jaeger: Diss. inaug., Berlin, 1876.—Schwenninger: Berl. klin. Woch., 1876, Nr. 32.—Münster: Dtsch. med. Woch., 1877, Nr. 14 and 15.—Delore: Ann. de Gyn., Feb., 1878.

[*] Berl. klin. Woch., 1874, Nr. 13; Berl. Beitr. z. Geb. u. Gyn., III., S. 71.

[†] Med. Times, Mar. 23d, 1872.

MYOMOTOMY (REMOVAL OF MYOMATA BY LAPAROTOMY).

Myomotomy varies a good deal according to the connections of the tumor.

Myomotomy in pediculated subperitoneal tumors.—The tumor is brought out through an abdominal incision made in the ordinary way. Where the growth is very large, *Schröder* thinks it better to extend the wound from the symphysis close up to the ensiform cartilage if need be, rather than to reduce the tumor gradually by "morcellement," as recommended by *Péan*. The pedicle is to be dealt with like that of an ovarian cyst. He prefers the intraperitoneal method. When there are large vessels he transfixes the pedicle, ties it on each side, and cuts the tumor off at about 2 cm. from the ligature. If the pedicle is not very vascular, he divides it by a wedge-shaped incision extending into the uterine wall, and brings the flaps together with deep sutures. Many small subserous polypi with thin pedicles may simply be enucleated with the fingers, the opening in the peritonæum being then closed with sutures.

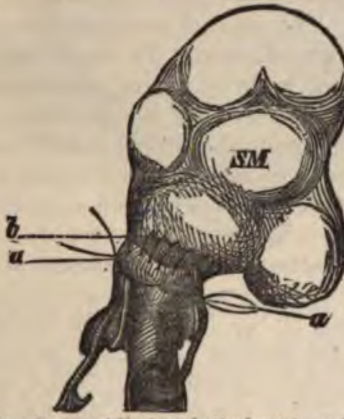


FIG. 14.—Myomotomy in a tumor with a broad attachment. *a*, ligature around the broad pedicle ; *b*, abscision of the tumor, together with the suture.

Myomotomy in tumors attached to the body of the uterus by a broad base.—Instead of performing supra-vaginal amputation, it is better to separate the tumor from the uterus in the manner shown in Fig. 14. Somewhat below the insertion of the myoma, a double-threaded needle is passed through the uterus, and the ligature is tied on each side (*a*) ; the tumor is then sliced out from the uterus as one would cut a piece out of a melon (*b*), and the two cut surfaces are then stitched together. The stump may be left tolerably large without danger of gangrene, provided no infectious material has gained access to the peritoneal cavity.

Myomotomy in interstitial tumors.—If a large myoma is situated wholly below the insertion of the Fallopian tubes, the folds of the broad ligaments will be separated, and the tumor can be removed only in common with the body of the uterus by amputation at the os internum. *Péan* elaborated a special method. Two long, strong needles are passed through the cervix, at right angles to each other, at the level of the os internum; a double wire is carried through midway between the needles; the cervix and the broad ligaments are then constricted in the wire loop on either side by means of a Cintrat's *serre-nœud.* The tumor is cut off, and the stump is fixed in the lower angle of the wound. In these cases, too, *Schröder* prefers the intra-peritoneal method of dealing with the stump. He first ties the broad ligaments with a double ligature (Fig. 15), including the ovaries; then cuts the broad ligaments between the ligatures, thus freeing the cervix. He now ties or constricts the cervix with a double ligature, and above the ligature he cuts off the tumor with a large, pointed knife; after which he fashions the stump into a wedge-shaped cleft, the sides of which he brings together from before backward with deep and superficial sutures.

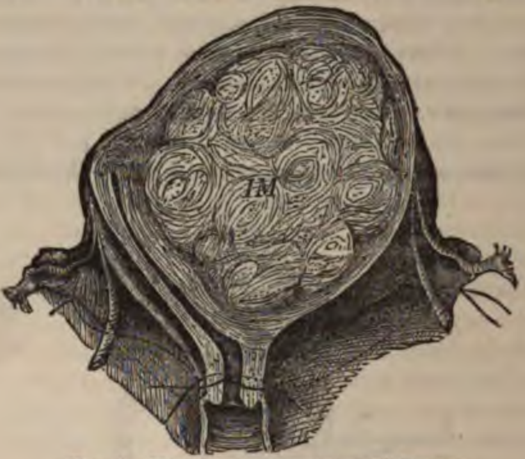


FIG. 15.—Myomotomy for an interstitial tumor.

When the tumor projects deep into a dilated cervix, laparotomy is contra-indicated—at least, total extirpation, as in Freund's method for cancer, would be too difficult and dangerous to be thought of. Neither is the operation generally allowable when a subserous myoma extends between the folds of the broad ligament or beneath Douglas's space, for its enucleation from the pelvic cellular tissue would involve the greatest dangers.

PROGNOSIS AND INDICATIONS OF MYOMOTOMY.

These matters cannot yet be settled by statistics. *Péan*[*] had only

---

[*] See POZZI, l. c.

8 deaths in 24 operations by his method; *Spencer Wells*,* 15 in 25; *Billroth*, 10 in 15; *Kœberlé*, 5 in 8; and *Schröder*, 7 in 18. The prognosis varies with the manner in which the tumor is attached to the uterus. Where the pedicle is slender, the prognosis is but little worse than that of ovariotomy, but the dangers of deep excision of the enlarged body of the uterus are far greater. The scope of the operation should at present be limited, but we may hope that the strictest observance of the antiseptic method, together with improved methods of procedure, may extend its range somewhat.

Among the *indications*, uncontrollable growth of the tumor stands first. Hæmorrhage, too, may be so profuse, so persistent, and so beyond our power to restrain, that the only hope may lie in laparotomy. The chances are worse, however, in very anæmic patients. Myomotomy is justifiable, in general, when the tumors irritate the peritonæum so as to produce ascites. Unbearable pain also may justify the operation. It is very rare for myomata capable of removal by laparotomy to get so wedged into Douglas's space that symptoms of incarceration call for laparotomy.

It has lately been attempted to check the growth of myomata, and possibly bring about their retrogression, with the result of stopping the hemorrhages, by *extirpation of the normal or but slightly diseased ovaries* (*normal ovariotomy, Battey's operation, castration*).† The idea is to produce an artificial menopause. *Trenholme* first removed the normal ovaries on this theory, then *Hegar* and others. In general, an incision is made through the linea alba, and then by twisting the tumor, first one ovary and then the other is reached. A double ligature is then passed through the mesovarium, and tied on each side. The ovary is then cut off. As the ovaries often lie in abnormal situations in these cases, thorough palpation should be practised before the operation. If one of them lies very far to the side, and the mobility of the tumor is limited, we may cut down directly upon the ovary. Adhesions may give rise to much difficulty. From the danger of the operation and its uncertainty, owing to our defective knowledge of the relations between ovulation and menstruation, its scope is still undecided. In one case *Schröder* saw an immediate improvement in the symptoms, with manifest softening and diminution of the tumor, but after a few months the hemorrhages returned, and the tumor began to grow again. He is inclined to think that the improvement may be due, not to the removal of the ovaries, but to the ligation of large vessels supplying the tumor. He has seen a fibroma, that could not be removed, almost disappear after tying large vessels in its omental adhesions. Until more convincing experience has accumulated, he would, therefore, restrict the operation to cases in which the symptoms threaten life, and it seems impracticable to remove the tumor, and at the same time would try to tie other large afferent vessels.

---

* Volkmann's Samml. klin. Vortr., 149–150, and Brit. Med. Jour., 1878, 12, 14.

† In 1872 this operation was first done; at about the same time, but for different purposes, by *Hegar* and *Battey*. HEGAR: Centralbl. f. Gyn., 1877, Nr. 17, u. 1878, Nr. 2; Wien. med. Woch., 1878, Nr. 15; Samml. klin. Verh., 136–138.—BATTEY: Trans. Am. Gyn. Soc., 1877, p. 101.—TRENHOLME: Am. Jour. of Obstet., IX., p. 703.—SIMS: Med. Times and Gaz., Nov. 24th, 1877.—BŒRNER: Dtsch. med. Woch., 1878, Nr. 47-50.—BEIGEL: Wien. med. Woch., 1878, Nr. 7 u. 8.—TAUFFER: Pester med.-chir. Presse, S.-A.—MARTIN, A.: Berl. klin. Woch., 1878, Nr. 15 u. 16.—AVELING: Obstet. Jour. of Gt. Brit. and Ire., Jan., 1878.

ADENOMA OF THE UTERUS.

We meet with growths from the endometrium which, as they are manifestly made of newly-formed glandular tissue, we must call adenomatous. They occur in various forms.

1. *Diffuse adenoma* closely resembles the endometritis fungosa of *Olshausen*, differing from it only in being almost wholly made up of glands. There is no cystic degeneration of the newly-formed glands, for their lumen is but slightly, if at all enlarged. There seem to be transitions between fungous endometritis and diffuse adenoma, and between the latter and the normal condition. As a rule, however, either fungous endometritis is the prominent element, with small-celled proliferation of the interglandular connective tissue, or else the newly-formed glands prevail, so that in extreme cases, thickly-tangled masses of gland-ducts are seen with the microscope. These latter cases must be reckoned as examples of malignant adenoma (Fig. 16). It would seem, too, that adenoma is somewhat frequently transformed into true glandular carcinoma, the cylindrical epithelia of the glands being increased in amount, and the lumina becoming clogged with atypical forms of epithelial cells.

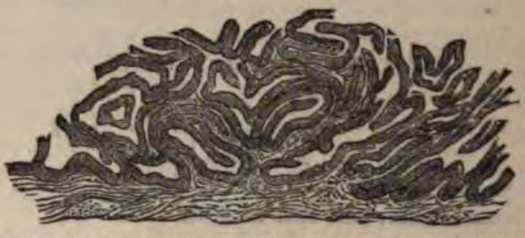


FIG. 16.—Malignant adenoma of the uterine mucous membrane.

2. Adenoma may occur in the form of polypi, either by the massing of diffuse growths, or by individual formation. In still other cases, the adenomatous polypus has a firm fibrous pedicle, with normal and cystically degenerated glands seated upon its branches.

ETIOLOGY.

The disease occurs most commonly, but not exclusively, about the time of the menopause, or later still, in nulliparæ and others indifferently. It can only in exceptional cases be traced to irritations of the endometrium, and in most cases of that kind, the form is not typical, but rather bordering upon fungous endometritis, which may follow chronic catarrh or abortion. The invasion of pure diffuse adenoma, often many years after the climacteric, reminds one forcibly of the development of carcinoma of the body of the uterus, and renders it all the more probable that the latter is usually preceded by adenoma.

SYMPTOMS.

The only constant symptom is hemorrhage, sometimes as a menorrhagia, but more often irregular. It is more apt to be lingering than profuse. Frequently for months or years there are no other symptoms; in particular, leucorrhœa may be trifling or altogether wanting, although it is more marked in some cases, especially those of malignant adenoma. As a rule, there is no pronounced pain, although there may be sensations of pressure and fulness within the pelvis, or bearing-down pain in the sacral region, and manifold nervous symptoms, which latter are generally due to anæmia. There is no special cachexia; on the contrary, although the mucous membranes may look anæmic (as in patients with myomata), there may be an abundant development of fat.

DIAGNOSIS.

Obstinate hemorrhages should suggest the presence of growths from the endometrium. If the patient is of advanced age, if the hemorrhages have come on gradually without ascertainable cause, and if there is not much discharge, the probability of an adenomatous condition of the mucous membrane is increased. By ordinary examination, we simply find the uterus moderately enlarged, soft, and somewhat tender. To establish the diagnosis, pieces of the mucous membrane must be examined microscopically. By using a slender curette, we need not dilate the cervical canal. In cases of adenomatous polypus, we may at least easily make out that there is a polypus.

PROGNOSIS.

The more the outgrowths consist of newly-formed glands, and the longer the disease has lasted, the more serious is the prospect. Treatment may control the symptoms for months together, but they gradually return, rendering removal of the growth necessary, until finally, perhaps years afterward, pronounced glandular carcinoma is developed in the uterine cavity. It seems that the prognosis is always good in cases of circumscribed, true adenomatous polypus.

TREATMENT.

Adenomatous polypi should be treated like other polypi. In the diffuse form, the whole inner surface of the uterus must be thoroughly scraped, which, with a small curette, can generally be done without dilating the cervix. The bleeding is moderate, and the result never fails. Where the glandular elements are profuse, it is well to cauterize the scraped surface with injections of chloride of iron or tincture of iodine, the cervix being first dilated. Hoffmann's syringe may be used without dilating. *Schröder* considers it free from danger. Perhaps in the future it will be found justifiable to extirpate the uterus for marked diffuse adenoma, so close does it approach to malignancy.

CANCER OF THE CERVIX UTERI.

TREATMENT.

Cases of extension to the fundus seemed not to admit of operation until Freund brought forward his method of *total extirpation of the uterus by laparotomy.*[*] The operation is performed in the following manner:[†] After thoroughly disinfecting the whole ulcerated area and the vagina with strong solutions of carbolic acid, the abdominal incision is made under the strictest antiseptic precautions. It should extend down to the symphysis, and the skin may be cut a little lower. Unless the recti are unusually yielding, their attachment to the symphysis is cut through. The intestines are now lifted out of Douglas' space, and carried to the upper portion of the abdomen, or, if (as is generally the case) this cannot be done, they are wrapped in a warm carbolized cloth. By dragging the uterus upward, the broad ligaments are so put upon the

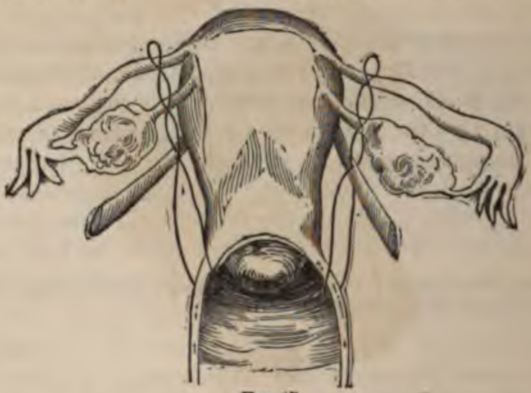


Fig. 17.

---

[*] W. A. FREUND: Volkmann's Vortr., Nr. 133; Centralbl. f. Gyn., Nr. 12; Berl. klin. Wochensch., 1878, Nr. 27.—FRAENKEL: Berl. klin. Woch., 1878, Nr. 31.—Tageblatt der Casseler Naturforschervers., S. 17.—BERNS: Nederl. Tijdschr. voor Geneesk., 1878, No. 15.—MASSARI, J. v.: Wien. med. Presse, 1878, Nr. 45 u. 46; Centralbl. für Gyn., 1879, No. 11.—KOCKS: Arch. f. Gyn., XIV., S. 127.—CREDÉ: *Ibid.*, S. 430.—LOEBKER: *Ibid.*, S. 460.—BRUNTZEL: *Ibid.*, S. 425.

[†] I describe the method employed by *Schröder*, which, in all essential points, corresponds with *Freund's*.

stretch as easily to be tied. This may be done in Freund's way, with three loops (one reaching from the tube to the ovarian ligament, one from the latter to the round ligament, and one from the round ligament to the vault of the vagina) (see Fig. 17), or the two upper loops may be replaced by a single one, embracing both the tube and the ovarian ligament. The ligature to encircle the vault of the vagina is carefully applied by means of a peculiar needle devised by Freund. Only a small portion of the vaginal mucous membrane is to be included in the loop, otherwise it cannot be drawn tight enough. The anterior and posterior vaults of the vagina are then cut through. Anteriorly, the peritonæum is freely cut across in the vesico-uterine excavation, the bladder is cut clean away from the uterus, and the vaginal wall incised. The posterior wall gives little trouble. The broad ligaments are now divided between the ligatures and the uterus, and the whole organ is then free from its attachments. If the uterine arteries spout, they should rapidly be tied or twisted. The great wound remaining is then closed from before backward with "*ligatures perdues*" cut short, the first penetrating the anterior fold of the peritonæum, then traversing the retro-vesical cellular tissue, and finally emerging from the anterior wall of the vagina. The posterior lip of the wound is formed by the posterior vaginal wall and the peritonæum of Douglas's space. The whole is closed in such manner as to be covered everywhere with peritonæum. The intestine is then returned to the pelvis, and the abdominal cavity is closed. The operation is very difficult and dangerous. It is indicated only when all the diseased parts cannot be removed by supra-vaginal excision. Such cases are very rare. When the cellular tissue of the pelvis has been invaded, no operation is to be thought of.

NORMAL MENSTRUATION.

The researches of *Kundrath* and *Engelmann*,* *Williams*,† *Leopold*,‡ and *Wyder*,§ although rather discordant, have overturned the theory of menstruation previously held. Whilst *Kundrath* and *Williams* look upon fatty degeneration of the upper layers of the mucous membrane as the cause of menstruation, *Williams* indeed even supposing that the entire mucous membrane is destroyed and renewed within the period of four weeks, *Leopold* and *Wyder* deny the occurrence of a primary fatty degeneration, and simply assume that a hemorrhage from the free surface and a superficial desquamation result from a stasis of blood in the upper layers. According to some researches by *Ruge*, yet unpublished, the latter is very doubtful. We may assume, at all events, that a certain retrogession of the mucous membrane, hypertrophied and rich in glands, accompanies the loss of blood, and that the occurrence of menstruation is to a certain extent a sign that the ovule of that period has perished unfertilized.

---

* *Stricker's* med. Jahrb., 1873, 2 Heft, S. 139.

† Obstet. Journ. of Gt. Brit. and Ire., Aug., 1874, p. 324; e. l., Nov., 1875, p. 496; Dec., 1875, p. 620; and Amer. Journ. of Obstet., VIII., p. 727 (See *Engelmann:* Am. Jour. of Obstet., VIII., p. 30.)

‡ Arch. f. Gyn., XI., S. 110.

§ *Ibid.*, XIII., S. 1.

CHRONIC OOPHORITIS.

PATHOLOGICAL ANATOMY.

But little is known of it. The ovaries are enlarged, but only moderately, as a rule. Slight cystic degeneration and interstitial inflammation were repeatedly found by *Beigel** in casual autopsies, and perhaps they may be regarded as the foundation of chronic oöphoritis. *De Sinéty*† likewise found slight cystic degenerations of the Graafian follicles, with inflammatory phenomena at the surface of the organ.

ETIOLOGY.

It is most common in ill-nourished young women who have generally been through parturition, and have had vaginal or uterine catarrh. It may be unilateral or bilateral; it is generally bilateral, but is most marked on one side. The tubes are generally somewhat swollen, or there are evidences of previous perimetritis—adhesions and false membranes. Gonorrhœa seems to be a frequent cause, the inflammation extending from the vagina to the endometrium, and thence, by way of the tube, to the ovary. The organ may be in its normal situation, but it often falls down deep into Douglas's space.

SYMPTOMS.

The pain is acute. Patients complain of a drawing pain in the lower part of the abdomen, especially on exertion, even standing and walking. In severe cases they cannot assume the erect posture without the greatest pain, which ceases only when they lie down again. Constipation notably increases the suffering, perhaps by the pressure of fæcal masses upon the prolapsed ovary. The necessary inactivity leads to loss of appetite and sleep, and the nutrition suffers, so that the patient presents a piteous spectacle, worn down with persistent pain and nervous disturbances. Menstruation is often irregular and abundant, and the characteristic pains are increased.

* Wien. med. Wochensch., 1878, Nr. 7 u. 8.

† Arch. de Physiol., 1878, No. 1.

DIAGNOSIS.

This cannot be positive unless swelling and tenderness of the ovary are made out by examination. With the organ in its normal situation, this is possible only by thorough combined palpation. If it is prolapsed, vaginal or rectal palpation is often sufficient. When but slightly enlarged, however, we can never examine the ovary by itself through the abdominal wall. In incipient cystic degeneration, the surface is nodular and there is no tenderness.

PROGNOSIS.

There is no danger to life, but it is difficult to foretell the duration of the disease. It is by no means incurable, however.

TREATMENT.

Rest in bed, abstinence from cohabitation, and attention to the bowels and bladder are of the first importance. If the pain is excessive, an ice-bladder is the best application, but otherwise the protracted use of the wet bandage or counter-irritation with tincture of iodine or a blister is to be advised. When some improvement has been accomplished, mild baths, either general or to the hips, may be taken, of a temperature of 27°-28° R., and then the general strength should be attended to. Amongst bathing resorts, Schlangenbad and Landeck have the best effect. Catarrhal affections should be cured. Where the genitals are much relaxed, the suffering is often materially mitigated by wearing a Mayer's ring-pessary, which diminishes the displacement of the parts that takes place on bearing-down efforts. "Normal ovariotomy" may fairly be resorted to in incurable cases where all the functions are seriously interfered with.

OVARIAN CYSTS.

DIAGNOSIS.

The co-existence of ovarian tumors and uterine myomata is no rarity. By thorough conjoint examination a positive diagnosis of this condition may generally be made, for we can distinguish the uterus, characteristically enlarged, from the elastic ovarian tumor connected with it by a pedicle.

In cases of large tumors there may be great difficulty in determining whether or not both ovaries are affected, and, if only one, which one; but practically the decision is not very important, and we must always be prepared to find the disease on both sides.

It may become important to diagnosticate *rupture, twisting of the pedicle,* and *malignant degeneration of the tumor.* In cases of small cysts, rupture causes no special symptoms. If symptoms of shock appear after the rupture of very large cysts, we should take torsion into account, for the symptoms may be almost the same. The latter is probable if the tumor has suddenly become enlarged, with severe pain and symptoms of peritonitis. Almost all cases of sudden general peritonitis with ovarian cysts are due to twisting of the pedicle. Occasionally we may feel the thick, hard, twisted pedicle, if the uterus be forced down.

An abundance of ascitic fluid speaks in favor of malignant degeneration. Especially is this the case when the papillary outgrowths from the ruptured cysts infect the peritonæum. They may often be recognized through Douglas's space, having a characteristic snowball-like feel on pressure. The fluid withdrawn by puncture will often settle the question, fragments of cells and crystals of cholestearin being found with the microscope.[*]

PERIMETRITIS.

TREATMENT.

If an abscess points externally, it should be freely opened, injected with carbolized water, a drain inserted, and antiseptically treated.[†] Sometimes very large cavities close up unexpectedly fast, and in other cases sinuses remain open for a long time. It is questionable if oöphorectomy would be justifiable to stop relapses.

THROMBUS OR HÆMATOMA OF THE CELLULAR TISSUE. (EXTRA-PERITONEAL HÆMATOCELE).

As a rule, these bloody effusions are seated in the broad ligaments, one or both; if in both, they are generally connected by an isthmus, that runs more commonly behind than in front of the uterus. It is exceptional for them to be larger than a hen's egg. If large, they may raise the uterus decidedly. They are of a doughy consistence. If tolerably large, besides the signs of anæmia, sudden pain occurs, which may be extremely severe and of a peculiar cramp-like character, owing to distention of the ligaments. They generally end in absorption. Their sudden onset without fever distinguishes them from inflammation, whilst their irregular shape and lateral situation mark them as extra-peritoneal. The treatment should generally be expectant, incision being indicated only when rupture into the peritoneal cavity is threatened.

---

[*] See Foulis: Brit. Med. Jour., June 26th, 1875, July 20th, Nov. 2d, 1878; and Knowsley Thornton: *Ibid.*, Sept. 7th, 1878.

[†] Kaiser: Dtsch. Arch. f. klin. Med., XVII., S. 74.—Buch: Charitéannalen, IV., S. 360. Berlin, 1879.

TUMORS OF THE PELVIC PERITONÆUM AND CELLULAR TISSUE.

Ecchinococci may occur in the most various situations. The symptoms are seldom very characteristic, denoting only a tumor. They have generally come under notice as causes of dystocia. A positive diagnosis can be made only after their rupture into the vagina or the rectum, or after puncture. Without operation or the occurrence of inflammation consequent on rupture, the prognosis is unfavorable. Incision and destruction of the sac are most to be recommended. *Schröder* once successfully removed the entire sac, supposing it to be an ovarian tumor.

INFLAMMATION OF THE VAGINA.

PATHOLOGICAL ANATOMY.

The swelling of the mucous membrane shows marked peculiarities during pregnancy.* The folds swell, forming hump-like projections, separated by deep fissures. Cystic spaces form in the ridges, containing

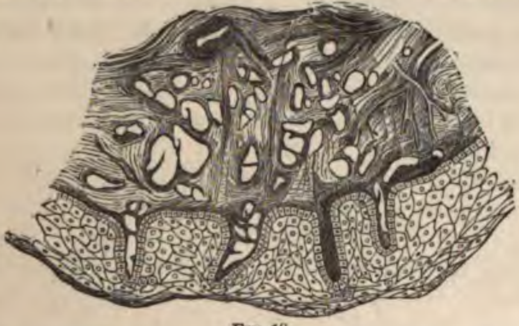


FIG. 18.

---

* WINCKEL: Arch. f. Gyn., II., S. 406.—SCHRÖDER: Dtsch. Arch. f. klin. Med., 1874, S. 538.—SCHMELLING: Diss. inaug. Berlin, 1875.—NAECKE: Arch. f. Gyn., IX., S. 461.—CHENEVIÈRE: *Ibid.*, XI., S. 351.—ZWEIFEL: *Ibid.*, XII., S. 39.—RUGE, C.: Ztschr. f. Geb. u. Gyn., II., S. 29.

fluid and gas, so that the vagina seems coated with a mass of cysts. This form rarely occurs except during pregnancy. *Winckel*, who first called attention to it, called it *colpohyperplasia cystica*. According to *C. Ruge*, the air occupies clefts in the cellular tissue, and the affection is therefore better denominated *colpitis emphysematosa*. How the gas gets there we do not know. (See Fig. 18.)

SYMPTOMS.

Beyond a certain amount of discharge, emphysematous colpitis gives so little trouble that its discovery is generally accidental.

PROGNOSIS.

Colpitis is very obstinate, unless due to mechanical irritation that can be removed, like that of a pessary. The gonorrhœal form may improve with or without treatment, but relapses are exceedingly apt to occur, although usually due to renewed cohabitation with the author of the infection. Senile colpitis is a trifling affection, and the partial agglutinations caused by it do no harm.

TREATMENT.

The causes should first be removed. In chlorotics, iron will often stop the discharge without local treatment. If there is prolapse, a proper ring-pessary will frequently affect a cessation or marked decrease of the secretion. Any co-existing uterine disease, especially cervical catarrh, will need attention. In the local treatment, mere cleanliness often suffices, since the stagnant secretion is in itself a source of irritation. The necessary injections should be very carefully managed, the instrument being gently introduced, avoiding too strong a stream, and using the water neither too warm nor too cold—about 28° R. at first, gradually lowered. If cold injections are not well borne, we should desist from them. Injections of alkaline-spring waters, such as those of Ems and Neuenahr, are particularly effective. Chronic cases, however, resist these simple measures, and we must then use astringents—pyroligneous acid, tannin, alum, chloride of iron, nitrate of silver, and the like. When these are indicated, it is better to pour the solution in through a cylindrical speculum, which is then slowly withdrawn so as to bring the liquid in contact with every portion of the vagina. This is better than painting the mucous membrane through a speculum. Small tampons or pieces of sponge soaked in astringents may be used. *Scanzoni* recommends four parts of tannin to thirty of glycerin. Glycerin alone does well, acting as a depletive by virtue of the serous discharge that it sets up. The tampons may be sprinkled with powdered alum, or smeared with alum ointment (5 to 30, according to *Hildebrandt*). Tannin suppositories (1 part of tannin to 4 of cocoa butter) also answer well. In gonorrhœal colpitis, strong solutions of carbolic acid are most to be commended (as strong as 5 per cent), poured in through a speculum; any lingering discharge being treated with pyroligneous-acid injections.

The last-named agent answers best in senile colpitis, irritating the cells of the rete Malpighii to the increased production of flat epithelia.

URO-GENITAL FISTULÆ.

TREATMENT.

Bozeman lays special stress upon preparatory treatment of the fistula, consisting essentially in incision and stretching of cicatrices and in dilatation of the vagina with large hard-rubber balls, so as to make the edges of the fistula accessible. After refreshing the edges, with the patient fixed in the knee-elbow posture by means of a special apparatus, he closes the fistula with a peculiar suture, in which the silver wire passes through a plate of lead lying directly upon the wound, and is clamped with perforated shot.

The *closure of fistulæ of the ureter* involves great difficulties. *Landau*[*] introduces a slender catheter into the upper part of the ureter from the vagina, and then passes the free end of the catheter through the vesical portion of the ureter into the bladder and out through the urethra. The tissue is now refreshed on either side of the catheter, and brought together over it.

VAGINISMUS.

Pain in copulation (the *dyspareunia* of Barnes), resulting from diseases within the pelvis, should not be confounded with vaginismus. When, however, the uterus is low and fixed by inflammatory adhesions, such pain may be caused by the impingement of the penis, that spasmodic action, quite like that of vaginismus, will occur.

LACERATIONS OF THE PERINÆUM.

Simon's operation is varied by some surgeons. *Hildebrandt*[†] uses deep perineal sutures, embracing the whole wound. *Freund*[‡] leaves the

---

[*] Arch. f. Gyn., IX., S. 426.

[†] Handb. d. Frauenkr., S. 88.

[‡] *Ibid.*, S. 95.

mna of the vagina intact, and refreshes on either side of it.
r, but with sliding of flaps, *Bischoff* performs colpoperineo-
o lapsus.

PRURITUS VULVÆ.

the pruritus is a symptom of diabetes, a course at Carlsbad
st chance of cure.

DISEASES OF THE
PERIPHERAL CEREBRO-SPINAL NERVES.

BY

S. G. WEBBER, M.D.,

OF BOSTON,

CLINICAL INSTRUCTOR IN DISEASES OF THE NERVOUS SYSTEM, HARVARD UNIVERSITY; VISITING PHYSICIAN TO DEPARTMENT FOR DISEASES OF NERVOUS SYSTEM, CITY HOSPITAL, BOSTON;
CONSULTING PHYSICIAN, CARNEY HOSPITAL, BOSTON.

NEURALGIA.

BIBLIOGRAPHY.

PROF. VERNEUIL: Des Nevralgies Traumatiques Secondaires Précoces. Arch. Gén. de Méd., 1874, pp. 528, 679.—GEO. H. EVANS: Nitrite of Amyl in Facial Neuralgia. Practitioner, Sept., 1875, p. 179.—PROF. V. PITHA: On the Diagnosis and Treatment of Neuralgia, especially by Hypodermic Injection of Morphia, Founded on Personal Experience. Med. Times and Gaz., Sept. 25th and Nov. 27th, 1875, pp. 356, 591.—GEO. W. BALFOUR: On Obstinate Trigeminal Neuralgia as a Symptom of Syphilitic Cerebral Disease, its Diagnosis and Treatment. Edinburgh Med. Jour., Oct., 1875, p. 290.—DAVID YOUNG: Two Cases of Neuralgia treated with Chloride of Ammonium. Practitioner, Dec., 1875, p. 412.—HERMANN SCHULZ: Die Subcutane Carbolsäureinjection als Anästheticum und Antineuralgicum. All. med. Cent. Zeit., 61, 1875.—C. LANGE: Ueber Neuralgieen und deren Behandlung. Hosp. Tidende, 2, R. 11, 1875 (Abstract in Schmidt's Jahr., 169, p. 240).—O. BERGER: Zur physiologischen und therapeutischen Würdigung des Gelsemium sempervirens. Centralbl. f. d. m. Wissen., 2. Oct., No. 43, 1875, p. 721, et seq.—A. JURASZ: Gelsemium sempervirens als antineuralgisches Mittel. Centralbl. f. d. med. Wissen., 31, 1875, p. 513.—S. W. MITCHELL: Neurotomy. With an Examination of the Regenerated Nerves and Notes upon Neural Repairs by R. M. BERTOLET. Am. Jour. Med. Sci., April, 1876, p. 321.—CHARLES S. BULL: The Influence of Neuralgia of the Trigeminus in causing Iritis and Choroiditis and its Pathological Significance. N. Y. Med. Record, Aug. 19th, 1876, p. 535.—E. P. HURD: A Contribution to the Study of Neuralgia. An Intractable Case in Private Practice. Med. Record, Nov. 18th, 1876, p. 743.—E. MARKHAM SKERRITT: Croton Chloral in Neuralgia. Lancet, Dec. 2d, 1876, p. 776.—USPENSKY: Versuch einer Pathologie der Neuralgien. Deut. Arch. f. kl. Med., XVIII., 1876, p. 36.—JULIUS SCHIFF, JR.: Drei Fälle von Neuralgia Facialis, verursacht durch Dentinneubildung in der Pulpahöhle. All. Wien. med. Zeit., 1876, 24 and 26.—PATRUBAN: Die Unterbindung der Carotis communis als letztes Heilmittel des Tic douloureux. All. Wien. med. Zeit., 1876, Nos. 48, 49, 50.—MOOS: Ueber den Zusammenhang zwischen Krankheiten des Gehörorgans und solchen des Nervus Trigeminus. Virch. Arch., LXVIII., 1876, p. 433.—A. SEELIGMÜLLER: Ein Fall von einer auf den Nervus Cutaneus Brachii Internus minor beschränkten Neuralgie. Arch. f. Psych. u. Nervenk., VI., 1876, p. 575.—C. S. BULL: A Case of Hysterical Hemiplegia with Neuralgia of the Trigeminus and Amblyopia, occurring in a family of marked Neurotic Characteristics. Med. Record, Feb. 17th, 1877, p. 99.—WEINLECHNER: Zur Statistik der Nervenresektion, Wien. med. Presse, 4, 1877, p. 116.—VICTOR URBANTSCHITSCH: Zur therapeutischen Verwendung des Amylnitrat. Wien. med. Presse, XVIII., 1877, Nos. 8, 9, 10, 11.—J. S. JEWELL: Pathology and Treatment of Neuralgia. Journal of Nervous and Mental Diseases, April, 1877, p. 207.—S. W. MITCHELL: The Relations of Pain to Weather: being a Study of the Natural History of a Case of Traumatic Neuralgia. Am. Jour. Med. Sci., April, 1877, p. 305.—E. M. BODDY: Neuralgia and its Treatment. Med. Times and Gaz., 6th Oct., 1877, p. 383.—ERSKINE MASON: A Case of Neuralgia of the Second Metatarso-Phalangeal Articulation Cured by Resection of the

Joint. Am. Journ. Med. Sci., Oct., 1877, p. 445.—A. C. WOOD: The Trigeminal Neuralgias. Med. Record, Oct. 27th, 1877, p. 673.—JULIUS SCHREIBER: Ueber Occipital-Neuralgie. Berlin. kl. Wochen., XIV., 1877, 50, p. 726.—EYMERY-HEROGNELLE: Etude du Gelsemium sempervirens et de son Action dans le Traitement des Neuralgies. Thèse de Paris, 1877.—GEO. LAWSON: Surgical Treatment of Neuralgia. Med. Times and Gaz., Feb. 9th, 1878, p. 137.—CARL FIEBER: Drei Fälle von Gesichtsneuralgie durch Nervenresection geheilt oder gebessert. Berlin. kl. Wochensch., 19, 1878, p. 269.—CH. FERNET: De la Sciatique et de sa Natur. Arch. Gén. de Méd., Avril, 1878, p. 385.—FÉRÉOL: Tic Douloureux de la Face d'Origin Epileptique, de son Traitement par le Sulfate de Cuivre Ammoniacal. Gaz. Hebd., 19th July, 1878, p. 465.—ABBOT: Salicylic Acid in Neuralgia. Boston Med. and Surg. Jour., Oct. 17th, 1878, p. 500.—E. C. SEGUIN: Report of Committee on Neurotics to Therapeutical Society, N. Y., on Aconitia. New York Med. Jour., Dec., 1878, p. 621.—ANGELÉ: Contribution à l'étude de Nevralgies au Point de Vue de leur Natur et de leur Traitement par les Injections irritantes. Dissert., Paris, 1878.—E. C. SEGUIN: A Contribution to the Medicinal Treatment of chronic Trigeminal Neuralgia. Med. Record, Jan. 4th, 1879, p. 6.—WALTER RIVINGTON: Excision of a Portion of the Spinal Accessory Nerve. Brit. Med. Journ., Feb. 8th, 1879, p. 212.—PROF. MASSINI: Gelsemium in Neuralgia. Dublin Jour. Med. Sci., May, 1879, p. 415.—ARMIGAND: Du Point Apophysaire dans les Névralgies et de l'Irritation Spéciale. Jour. de Thérapeut., 1879, p. 395.—R. W. TAYLOR: Clinical Notes on Neuralgia of the Sciatic Nerve Caused by Syphilis. N. Y. Med. Jour., Mar., 1880, p. 235.

Attempts are still made to explain the nature of neuralgia. It is scarcely worth while to notice mere definitions, but there have been more elaborate efforts made by *Uspensky*, *Jewell* and others. *Uspensky* considers that the change in the nervous system which depends upon the sense of pain most probably remains always the same, and is not modified by the peculiar nature of the irritation. Every pain is either caused by or at least accompanied with the formation of a certain quantity of the products of metamorphosis in the nerve tissues and by the action of this product upon the nervous system. This product is acid, and the pain will disappear only after the neutralization of this acid by the alkali of the blood and its subsequent absorption.

He explains the periodicity of neuralgia thus: the production of this acid irritant is continuous, but pain arises only when it is present in excess; if absorbed as fast as produced, there is no pain; hence, if too rapidly produced, or if the absorbing power of the blood-vessels is diminished, this irritant accumulates. Hence weakened function of the vasomotor nerves is one element in the etiology of neuralgia.

Every neuralgia is based upon irritation of the central terminal apparatus of the affected nerve, and the pathological changes are to be sought there, especially in a change in the size of the blood-vessels. In course of time the resorption of the irritant is diminished, as the result of previous overaction, and then it accumulates, until finally there is first tetanic vascular contraction and later paralytic enlargement. This disturbance of circulation, frequently repeated, may give rise to atrophy of the sensory nerve-cells and change in the calibre of the vessels with loss of their tone.

Dr. *Jewell* refers neuralgia to a lesion of the central nervous system

—the sensory tract, lying within the confines of the gray matter of the cerebro-spinal axis. Its essential seat is not in the peripheral nerves. The essential morbid condition in a neuralgia he claims to be a nutritive lesion of the central apparatus of cells which are the seats of true nervous sensibility. This state is frequently caused by disease of the peripheral nerves, but even in such cases the more irritable state of the sensory tract is the main factor. In this condition it reacts with pain to even trivial impressions made on the sensory nerves, which terminate in the affected region. A nerve-fibre cannot be brought into such a structural state by disease as to augment its conductibility beyond what is natural to it in a condition of health.

The attack of pain may be due to over-excitation, and hence overwear and waste of the affected centre, produced through the channel of its sensory nerves, or by changes in blood pressure in the affected centre, caused by loss or increase of tonus of the peripheral vessels, or a change in cardiac action, or by changes of posture, or of temperature, or of barometrical pressure, or by influences acting on the vaso-motor nerves, distributed to the diseased centres, and which may be affected from either the peripheral nervous system or from the cortex cerebri.

Lange considers that projected pain, or pain referred to the periphery, may originate either in the cord or in the peripheral nerves, not in the brain, except by direct influence of the peripheral nerve-fibres (unless in case of trigeminal neuralgia). Projected dysæsthesia may occur in brain disease, but not projected pain. If with brain disease there is pain felt at a distance, it is indirectly caused by disturbance of nutrition, inflammation, etc. The nerve tracts from the brain to the cord are not irritable to painful impressions; even in disease of the cord, pain is projected only in those nerve tracts which arise from the point of disease; below this there is only projected dysæsthesia.

Irritation of centripetal nerve-fibres may be projected by the central cells to other nerves, hence reflex neuralgia; this is most likely to arise from irritation of nerves of vegetative organs. Reflex pain caused by irritation of the pharynx is most likely to be felt in the lower jaw, neck, shoulder, and upper part of the chest; irritation of the nerves of the heart is felt in the same places and also in the upper extremities, and the entire thorax, more severely on the left; irritation in the stomach is felt in the thorax, often the arms, in epigastrium and hypochondrium; irritation in the gall-bladder and ducts is felt in the right arm and right side of the thorax; irritation in the intestines is felt in the lower part of the body and lower extremities; in chronic kidney and uterine disease the pain is felt in the lower extremities, with uterine irritation more as arthralgia than as sciatica. When the male genitals are affected the pain is felt in the legs. Disease of the digestive organs most frequently gives rise to severe reflex pain. Uterine irritation also frequently causes reflex pain.

S. W. Mitchell reports a very interesting observation of the influence which the weather may exert upon the occurrence of attacks of neuralgia. The patient was a very intelligent, educated gentleman who suffered from attacks of neuralgia in the stump of an amputated limb. Excepting during the paroxysms he was free from pain. The attacks were more frequent in the spring and autumn than in summer and winter. Neither falling temperature nor rising was alone sufficient to cause an attack. By color test during December to February the ozone was at 1 only twice during 18 attacks; generally it was at 0, or at most ¼. When the atmospheric pressure diminished—during the fall of the barometer and before it was complete—an attack of neuralgic pain occurred, and this was the more likely to take place when the lessening pressure culminated in rain. Sometimes with marked, sudden, and decided falls in the barometer, the pain seemed no greater nor continued longer than in slighter depressions.

The intensity of the neuralgia did not seem proportionate to the amount of rain-fall. At the exterior of a storm-disturbance the pain was usually less severe and might be only just perceived. A storm, reinforced by another at an angle say of 90°, producing great eccentricities in the curves, did not seem to produce a corresponding intensity or duration of the neuralgia. Abruptness of the fall of the barometer did not seem to have much to do with causing the pain; nor was the length of the attack dependent on the length of the storm. An atmosphere surcharged with moisture may be looked upon as the next most favorable single condition for the production of neuralgia. The separate factors of storms, as lessened pressure, rising temperature, greater humidity, winds, seemed as a rule incompetent, when acting singly, to give rise to attacks of pain. Either then it is the combination which works the mischief, or there is, in times of storms, some as yet unknown agency productive of evil. Such an agent may be either electricity or magnetism. These were not studied for lack of instruments, but when the aurora was brilliant, in '67–'68, neuralgia was prevalent. The pain belt may surround the storm belt and extend beyond the storm.

Prof. *Verneuil* recognizes and describes a neuralgia secondary to traumatisms, which is intermittent in type, resists antiphlogistics and narcotics, but yields without difficulty to sulphate of quinine. After cessation of the pain immediately consequent upon the injury, at a variable length of time, the secondary pain sets in; it may be local, or felt at a distance from the wound, even in regions innervated by branches anatomically distinct. This secondary pain is less acute than the primary and more varied in character; it is neuralgic in character, has remissions, sometimes extends over a large surface, sometimes is circumscribed, pressure does not increase it, movements of the parts awake it or increase it; it begins suddenly, generally in the night, rarely in the morning; the duration of the attack is limited to four or five minutes. This pain appears usually towards the middle of the second week, rarely later than thirteen or fourteen days, often earlier.

Among the causes of neuralgia which are rarely met may be mentioned the new formation of dentine at the roots of teeth pressing upon the nerves (*Julius Schiff, Jr.*). *Balfour* looks upon obstinate trigeminal neuralgia as a symptom of syphilitic cerebral disease; he says if the disease can be localized about the sella turcica, the probability is great that it is syphilitic in character, so great, that we are justified in treating the patient accordingly. *Taylor* finds that sciatica, depending upon syphilis, may appear as soon as six months after the primary lesion. *Stevens* thinks that, among the centripetal influences which give rise to neuralgia, the irritation arising from a perplexity or exhaustion of nerves engaged in the function of accommodation of the eye must be regarded as by far the most frequent and important; and when the eye is relieved the neuralgia ceases.

Fernet refers all cases of sciatica to a neuritis; he gives the account of one case, with autopsy, which confirms this view. His directions for examining the nerve in the living patient are, to have the patient on his back, with thighs slightly flexed, and the legs flexed on the thighs, so that the feet will rest comfortably on the bed. All the muscles of the legs are to be relaxed. The sciatic nerves are then examined with the fingers, which are pressed deeply into the popliteal space, and run along in the interstices of the muscles to the sciatic notch, the palmar surface of the fingers turned toward the external part of the thigh, and their ends moved backwards and forwards from within outwards; the sciatic nerve is very clearly felt as a large cord. On the diseased side, the nerve is larger, harder, and more cylindrical than on the healthy side, and is not changed by pressure. This change in the form and consistency of the nerve is sometimes uniform throughout its whole tract, sometimes is more or less limited.

H. C. Wood, for convenience, divides trigeminal neuralgias into two classes: I., where the paroxysms come on regularly, but at distant intervals. These are mostly symptomatic of several varieties of cachexia: Malarial—regularity of the intervals and the history of the case aid in the diagnosis; this is mostly supra-orbital. Megrim—connected with disturbed menstruation, gastric, anæmic, chlorotic, or syphilitic. Rheumatic neuritis—toxic, lead, arsenic, etc. II., coming on in sharp paroxysms at short intervals, and generally a reflex indication of peripheral irritation or centric pressure; three varieties: *a*, tic douloureux; *b*, anæsthesia dolorosa, and *c*, tic. In *a*, there is generally both pain and spasm; cause, usually peripheral, as a decayed tooth, pressure of cicatrix; *b*, caused by central tumor or clot pressing on the sensory fibres, causing pain and anæsthesia, with sensation of pain referred to the periphery.

Schreiber mentions a case of occipital neuralgia, complicated with vaso-motor affection, which is rare in neuralgia of that region. There was redness of the conjunctiva, increased secretion of tears and of nasal mucus. He explains these phenomena by the relations between the occipital nerve and the superior cervical ganglion.

Seeligmüller reports a rare case of neuralgia in which the *nervus cutaneus brachii internus* (nervus cutaneus medialis, Wrisberg's nerve) alone was affected. The pain began in the axilla, was of a burning character, lasted half an hour to an hour, recurring every two to four weeks. After a year, the pain was also felt in the left arm and shoulder, beginning just above the olecranon, running up the posterior part of the arm to the axilla, and thence across the lower angle of the scapula. Also the pain at times shot from the shoulder into the left breast. There were no painful points.

TREATMENT.

Prof. v. *Pitha* gives a detailed account of his own suffering from neuralgia, and the benefit he obtained from the hypodermic use of morphia. The precautions which he urges in regard to this use of morphia are excellent, the chief of which is to use small doses in the beginning; he advises as small as one-tenth to one-twentieth of a grain, or even less.

Herman Schulz injects under the skin a solution of carbolic acid, two parts of the pure acid to one hundred of distilled water, using from one-half to two syringefuls, the syringe holding about one gramme (about ♏15) distilled water. He repeats this in six to eight hours if the pain renders it necessary, without regard to the feverishness which may result. Urine was never colored green. He has had only one abscess in two hundred cases, and that was probably caused by an accidental motion of the patient, causing the syringe to tear the tissues. He thinks this will often take the place of morphia injections.

Of internal remedies, *croton-chloral* has been somewhat prominently brought forward. *Skerritt* used it in one hundred and twenty patients, found it of the greatest benefit in facial neuralgia. He obtained the most marked benefit in young patients, especially in the headache of anæmic women and girls. In these he obtained either relief or cure in eighty-six per cent; about the climacteric, the success was about fifty per cent, while later the ratio rose again to sixty per cent. At the climacteric, bromide of potassium seemed more reliable. Where there were marked hysterical symptoms he had less success. It is necessary to continue the use of the drug for some time to obtain permanent relief. In cases of pain in other regions than the head, he has not had success in the use of croton-chloral.

Gelsemium has been used with success by Prof. *Massini* in eighty cases of trigeminal neuralgia. The drug, when its physiological action is obtained, causes redness of the conjunctiva, pain in the eyelids, contraction of the pupil, double vision, giddiness. When the dose is increased there may be slight ptosis, dilatation of the pupil, gaping, langor, pain in the limbs. The respiration is not affected. He gave of the tincture twenty minims every half hour, till three doses were taken; he never had occasion to use more than sixty minims. Gelsemium may be used thus several days in succession.

Jurasz has found this drug of use in other than trifacial neuralgia, having employed it with wonderful effect in cases of brachial and sciatic neuralgia, and in muscular rheumatism. He used doses of from five to twenty drops three times a day.

The *ammoniacal sulphate of copper* has been used by *Fériol*. In one case of epileptic tic douloureux, the attacks recurring almost without interruption 60 to 100 daily, the above drug was used in dose of .05 the first day, .10 the second day, causing nausea without vomiting. This was continued three days with the effect of stopping the attacks. He has also used it in six other cases, four of which were cured as if instantaneously. He used .10 to .15 a day, increasing even to .30, or even .50. The formula used is:

℞ Aqu. dist.................................100.
Syr. aurantii flor.......................... 30.
Cupri ammoniati.......................... .10 to .15
Take a tablespoonful at meals.

It is important to continue the medicine for ten to fifteen days.

Prof. *Gubler* says he has never met a case of neuralgia of the fifth nerve, even tic douloureux, which did not yield to *aconitia*. A case where resection gave only temporary improvement was cured by .005 aconitine ($\frac{1}{14}$ grain). *Hottot's* preparation of nitrate of aconitine is the best to use, $\frac{1}{140}$ grain which contains $\frac{1}{280}$ grain of aconitine is the proper dose to begin with; this may be increased to $\frac{1}{12}$ grain. Heart disease absolutely counterindicates its use.

E. C. Seguin uses *Duquesnel's* crystallized aconitia. He found the susceptibility of individuals varied enormously, one patient having been severely affected by $\frac{1}{200}$ grain, while another tolerated $\frac{1}{84}$ grain every three hours with no special symptom. On the average distinct physiological and therapeutic effects were obtained by giving $\frac{1}{100}$ grain three times a day. Of six cases of severe trigeminal neuralgia one, which was probably reflex from a decayed tooth, was not benefited; three cases, epileptic in form, were slightly or only temporarily relieved; two cases were cured, in one of these the neuralgia had existed for seven years with an interruption of ten months after resection of the affected nerve. The form of trigeminal neuralgia which can be most certainly benefited by aconitia is not yet sufficiently defined. The formula used is:

℞ Aconitiæ (Duquesnel's)........................... ......gr. $\frac{1}{10}$
Glycerinæ,
Alcohol.................................. ....āā ʒ i.
Aq. menth. pip..........................q. s. ut fl. ℥ ij.
M. One teaspoonful = about $\frac{1}{140}$ grain.
Sig. One teaspoonful two or three times a day on an empty stomach.

In some cases he used $\frac{1}{8}$ or even $\frac{1}{6}$ grain aconitia in this formula.

The effects obtained from a decided impression made by the drug are, paralysis of heart, directly and by way of the vagus, pulse reduced, arterial tension lowered; subjective sensations are numbness and tingling of skin and mucous membrane, especially in the hands and tongue, chilliness and faintness, and indefinable nervousness.

Nitrite of Amyl has been used by *Urbantschitsch* in thirty cases, twenty-one or twenty-two cured. Some of these were followed for two or three months, others were lost sight of; sometimes the pain returned after three months or more, sometimes it did not relieve after a relapse. Its effects were very uncertain.

Evans used the amyl in three cases with relief or cure; large doses were necessary.

Dr. *Abbot* used *salicylic acid* with success in a case of sciatica.

Of operative measures *Weinlechner tied the carotid* after resection had failed in a case of inframaxillary neuralgia. *Patruban* had one death from pyæmia in eight or nine cases. The operation was successful where resection had failed, but was not invariably so.

S. W. Mitchell has collated one hundred and twenty cases of neurotomy. The immediate result in ninty-six cases was total relief. In eight more or less pain was felt again within three weeks; this is probably too soon for regeneration of the nerve to have taken place. Twenty-five were still well at the close of the first year; but five of these relapsed within two years. Twenty-seven had neuralgia again within periods varying from one to eighteen months. In four there was permanent relief from secondary operations.

Dr. *Mitchell* reports a case with repair of the nerve in six months after excision of two and one-half inches. In a second case one inch was removed and repair was observed in less than a year.

NERVE STRETCHING.

BIBLIOGRAPHY.

Gärtner: Deut. Zeit. f. Chir., 1872, 1, p. 462.—**Patruban**: Centralbl. f. m. Wissen., 1873, p. 254.—**Nussbaum**: Die chir. Klinik München, 1875.—**G. W. Callender**: Case of Neuralgia treated by Nerve Stretching. Lancet, 26th June, 1875, p. 883.—**Alexandre Duvault**: De la Distension des Nerfs comme moyen thérapeutique. Thèse de Paris 1876.—**Nussbaum**: Klin. Mittheil., München, 1876. —**Paul Vogt**: Nervendehnung bei traumatischem Tetanus. Centralbl. f. Chirurgie, No. 40, Oct. 7th, 1876, p. 625.—**Ferd. Petersen**: Zur Nervendehnung. Centralbl. f. Chir., No. 49, 1876, p. 769.—**Paul Vogt**: Die Nervendehnung als Operation der chirurgischen Praxis. Leipzig, 1877.—**John Chiene**: Nerve Stretching in Sciatica. Practitioner, June, 1877, p. 417.—**Thomas G. Morton**: Two Cases of Nerve Stretching. Am. Jour. of Med. Sci., Jan., 1878, p. 150.—**Joseph Bell**: Nerve Stretching in Sciatica. Edinb. Med. Jour., XXIII., Oct., 1877, p. 308.—**Sam'l F. Farrar**: On Prof. Nussbaum's Operation of Nerve Stretching. Chicago Med. Journal and Examiner, XXXVI., March, 1878, p. 230.—**E. Watson**: On Nerve Stretching in Acute Traumatic Tetanus, with Two Cases. Lancet, 1878, I., p. 229.—**Johnson Symington**: Lancet, 1878, June 22d, p. 904.—**Alex. W. Macfarlane**: Case of Sciatica treated by Nerve Stretching. Lancet, July 6th, 1878, p. 6.—**Klammroth**: Fall von Tetanus traumaticus erfolglos mit Nervendehnung behandelt. Deut. med. Wochensch., No. 44, 1878.—**Tage Hansen**: 2 Tilfaelde af Nervestraeking af Nervus accessorius. Hospitals Tidende, 2 R. V. Nr. 45, 1878, p. 705.—**Masing**: St. Petersburger med. Wochensch., No. 34, 1878. —**Baum**: Mimischer Gesichtskrampf. Dehnung des Facialis. Heilung. Berlin.

kl. Wochenschr., 7. Oct., 1878, p. 595.—CHIENE: Edinb. Med. Jour., Nov., 1878, p. 464.—STRUCKMANN: Tre Tilfaelde af Nervestraeking. En Exstirpation af et Cystosarcoma Cranii. Hospitals Tidende, 2, R. V., Nr. 44, 1878, p. 689.—v. NUSSBAUM: Die Operationen einer Intercostal-Neuralgie. Aerztl. bair. Intel.-Bl., 1878, XXV., p. 558.—BYRD: A Case of Sciatica of Six Months' Duration cured by Stretching the Sciatic Nerve. N. Y. Med. Record, Sept. 28th, 1878.—A. H. MARCHAND: Sur la Distension Chirurgicale des Nerfs. Gaz. Hebd., 14, 1878, p. 209.—A. BLUM: De l'Elongation des Nerfs. Arch. Gén. de Méd., 1878.—DEAHNA: Beiträge zur Nervenchirurgie zusammengestellt von Dr. D. zu Stuttgart. Schmidt's Jahrb., 184, 1879, p. 48.—ANNANDALE: Case of Spasmodic Wryneck successfully treated by Division of the Spinal Accessory Nerve after Failure of Stretching. Lancet, April 19th, 1879, p. 555.—T. G. STEWART: Remarks on a Case of Epileptiform Neuralgia treated by Nerve Stretching. Brit. Med. Jour., May 31st, 1879.—CHARLES HIGGINS: Two Cases of Nerve Stretching. Brit. Med. Jour., June 14th, 1879, p. 893.—KOCHER: Ueber Nervendehnung bei Trigeminus Neuralgie. Cor.-Bl. f. schweiz. Aerzte, IX., 1879, p. 324.—PROF. SPENCE: Stretching of Sciatic, Digital, and Infra-orbital Nerves. Lancet, Feb. 14th, 1880, p. 249.—LUMNIEZER: Ueber Nervendehnung. All. med. Cent. Zeit., 24, 1880, p. 283.—J. A. ESTLANDER: Finska läkaresällsk handl., XX., 1878, p. 278. Schmidt's Jahrb., 184, 1880, p. 258.

Nerve stretching has only recently come into prominence as an operation for the relief of neuralgia and other nervous affections. As it has been used chiefly for neuralgia, it seems fitting to consider its use here separately for all nervous affections for which it has as yet been resorted to.

In 1869, *Billroth* exposed the sciatic in a young man who had epilepsy as the result of an injury. The object was to break up the adhesions of diseased nerves. The nerve was found healthy at the point exposed and was dissected out for some distance. It was necessarily stretched to some extent and the patient was benefited (see *A. H. Marchand*).

In 1872, *Nussbaum* performed a similar operation upon the nerves of the arm to relieve contraction with benefit to the patient. This operation by *Nussbaum* first led to similar operations for relief of spasm or pain, only instead of releasing the nerve from adhesions it is now forcibly stretched. *Gärtner* and *Patruban* were the next to perform this operation.

Any nerve which can be reached surgically may be stretched. As a fact the operation has been performed upon the facial, supra-orbital, infra-orbital, alveolar, mental, spinal accessory, the nerves of the brachial plexus, the musculo-cutaneous, median, ulnar, digital, intercostal, sciatic, crural, tibial. As has been mentioned, this operation has been resorted to most frequently to relieve pain, but it has also been tried as a remedy in tetanus, in other varieties of spasm as torticollis, blepharospasm, and in contractions, also in two cases of epilepsy apparently depending upon implication of a nerve in a cicatrix. *Blum* advises that the operation should be performed antiseptically, an incision being made parallel to the direction of the nerves, varying in length according to the distance of the nerve from the surface, generally 5 or 6 cm. is sufficient. If it is suspected that a foreign body is pressing upon the nerve, or if there is limited abnormal sensibility, the incision should be made at that point. The fingers or a grooved sound should be used to separate the nerve from surrounding parts. The nerve after being separated is to be raised upon the fingers, sound, or forceps, pulled strongly, according to the size of the nerve, in both directions. *Verneuil* advises to press the nerve between the fingers or thumb and the edge of the grooved director, so as to rupture the nerve-fibres, but *Blum* and others think this ought not to be done, as that is not simple stretching, but is equivalent also to division of the nerve and is unnecessary. After the stretching the nerve is laid back in its place, and the wound closed and dressed. The nerve may be so much longer than previously that it will necessarily take a curved or wavy position.

Callender states that nerves bear rough handling without injury if freed from their connections at the point where the traction is made; two or three inches of a large nerve may be exposed without injury to its nutrition. *Bell* mentions that on pulling the nerve a sensation was felt as if he was pulling a vegetable with long fibrous roots from the ground.

As to the amount of force which may be used, *Symington*, in a paper read before the Edinburgh Med. Chir. Soc. reports experiments made by himself upon the sciatic nerve of a dead subject, by attaching weights rapidly to it immediately below the gluteus muscle until the nerve broke. He found that 130 lbs. was the average weight required, the maximum being 176 lbs., the minimum 86 lbs. in a young woman who died of phthisis. *Tillaux* very nearly agrees with this. He found 54 to 58 kilogrammes (119 to 127.9 lbs.) necessary to rupture the sciatic; and 20 to 25 kilogrames (44.1 to 55.1 lbs.) to rupture the median and ulnar.

The immediate effect of stretching nerves varies much, perhaps depending partly upon the force used, partly upon the amount of crushing at the point where the force is employed. Thus the crushing recommended by *Verneuil* would cause paralysis. *Blum* says that simple stretching is not followed by motor or sensory paralysis. In some cases, however, those results have been seen, also there has been severe pain as the immediate result of the stretching, but in no case heretofore reported has there been mention made of permanent paralysis either of motion or sensation. There have been two deaths recorded after stretching a nerve for neuralgia, one by *Verneuil* from erysipelas; one by *Gärtner* from hemorrhage from the jugular vein, which had not been injured at the time of the operation.

In most cases of neuralgia the relief from pain was immediate. Of 29 cases of neuralgia thus treated, only two were not relieved; 3 were temporarily or only partially relieved. The remaining 24 were entirely relieved for longer and shorter periods, most up to the time the account was written. The longest date mentioned is 14 months, the shortest 1 month, and at those dates there had been no return of pain. These periods are not sufficient to establish definitively that there will be no return of pain, but the results do encourage the performance of the operation wherever neuralgia is peculiarly rebellious.

Blum thus sums up the value of the operation and indications for it: In cases of traumatic neuralgia success is complete, in non-traumatic the pain disappears, but success is less complete. Stretching is indicated in neuralgias which are rebellious to therapeutic agents, and clearly limited to one nervous department. It ought to be performed immediately when the neighboring nerves show a tendency to be invaded. In neuralgias of stumps, stretching should always be tried a short distance from the cicatrix.

In contraction and spasm the operation has been performed 8 times; in 6 there was recovery; in 1 no effect; in 1 the patient was only temporarily benefited. Three of these cases were cases of torticollis (two recoveries, one failure, which was subsequently benefited by resection). *Tage Hansen* reports two of these cases, both recovered; in one the head was turned to the left one hundred times in a minute, and this had continued four years. An incision was made at the posterior edge of the sterno-mastoid, the accessorius was exposed and stretched in both directions. When the effects of the anæsthetic had partially disappeared, the contraction recurred with moderate intensity; but ceased after about a quarter of an hour, and did not return. Active voluntary motion of the head was not interfered with. His second case was equally successful. *Hansen* remarks that, as the cause of the spasm is not known in every case, it may be central, and then a cure would not be certain.

In two cases of epilepsy, where the attacks were evidently due to irritation of the nerves in the leg or foot, stretching the sciatic gave relief.

Nerve stretching has also been used in seven cases of traumatic tetanus. Only two of these recovered; one only had partial relief, but died; four died without relief.

Blum thinks that the stretching acts by releasing the nerve from neighboring tissues which compress it, but its value is chiefly due to changes in structure and circulation; not only at the point where the operation is performed, but also at points more or less distant.

Vogt found that the centrifugal stretching had no effect upon the central organ, but the centripetal stretching was communicated to the peripheral distribution and acted on the terminal apparatus of the nerve. The anatomical changes consist in displacing and loosening the nerve in its sheath, combined with a greater tortuousnesss and enlargement of the vessels running to the nerves. Changes in the pressure and metamorphosis of tissue (dependent upon slowing of the circulation in the dilated vessels) are important factors in the change of function caused by stretching.

Davault says there have been found in animals twenty-five days after stretching a nerve a few degenerated fibres. In another case, thirty days after, no such degenerated fibres were found.

SPASM AND TETANY.

BIBLIOGRAPHY.

JOHN HADDON: On Tetany. Brit. Med. Jour., Oct. 9th, 1875, p. 456.—THEODOR LANGHANS: Zur Casuistik der Rückenmarks-Affection (Tetanie und Lepra Anæsthetica). Virch. Arch., LXIV., 1875, p. 169.—FRIEDRICH SCHULTZE: Linkseitiger Facialis-Krampf in Folge eines Aneurysma der Arteria Vertebralis Sinistra. Virch. Arch., LXV., p. 385.—S. W. MITCHELL: On Functional Spasms. Am. Jour. Med. Sci., Oct., 1876, p. 321.—FR. CHVOSTEK: Beitrag zur Tetanie. Wien. med. Presse., Nos. 37, 38, 1876.—THOMAS BUZZARD: A Case of Tetany. Practitioner, July, 1877, p. 11.—FR. CHVOSTEK: Weitere Beiträge zur Tetanie. Wien. med. Presse, 1878, 1879.—N. WEISS: Ueber einen letal abgelaufenen Fall von Tetanie. Wien. med. Presse, Feb. 22d, 1880, p. 245.

S. W. Mitchell gives the details of several unusual cases of functional spasm, the spasms being excited by voluntary motion of one set of muscles either in neighboring or distant muscles. The seat of the morbid process giving rise to these acts is uncertain. He concludes: "Voluntary acts give rise to spasms in the muscles willed to move, or in remote groups of muscles. There is at times an unusual discharge of nerve force in some of these cases, or else the muscle itself has become the means by its over-use of hypersensitizing the sensory centre, which takes record of its activities, so that from this centre at times excito-motor impressions are radiated on to near or remote centres and result thus in spasms. It will be found in all these cases that, when an ordinary functional motor act gives rise to spasms elsewhere, these occur in muscles which have physiological and therefore anatomical relations to the muscles which, by their normal use, gave rise to the morbid activities."

TETANY.

T. Buzzard reports a case in a child ten years old.

Chvostek has reported very many cases of tetany. His observations have confirmed the statements made by *Erb* in regard to the electro-contractility of the muscles in such cases, and completes them in regard to the facial.

Weiss reports a fatal case occurring after extirpation of the thyroid gland. At the autopsy the recurrent nerve was found implicated in the wound. There were a few small hæmorhages around vessels in the cord, else nothing.

VRITER'S CRAMP AND ALLIED AFFECTIONS.

BIBLIOGRAPHY.

G. V. Poore: Dancer's Cramp. Brit. Med. Jour., Nov. 20th, 1875, p. 640.—
rl Hertzka: Zur therapeutischen Würdigung des Gelsemium sempervirens.
ntral. f. d. med. Wissen., No. 47, 1875, p. 803.—Onimus: Crampes des Employés
Télégraphe. Gaz. méd. de Paris, 15, 1875.—W. R. Gowers: Remarks on Two
ses of Writer's Cramp. Med. Times and Gaz., Nov. 17th, 1877, p. 536.—G. V.
ore: An Analysis of Seventy-five cases of "Writer's Cramp" and Impaired
riting Power. Med. Chir. Trans., LXI.—L. Bianchi: A Contribution to the
eatment of the Professional Dyscinesiæ. Brit. Med. Jour., Jan. 19th, 1878.—
imus: Le Mal Télégraphique. Gaz. des. Hôp., No. 32, 1878.—Geo. M. Beard:
nclusions from the Study of One Hundred and Twenty-five Cases of Writer's
amp and Allied Affections. The Medical Record, March 15th, 1879.

G. V. Poore has carefully analyzed seventy-five cases of impaired writ-
g power, including cases of paralysis, of writer's cramp and of cramp or
asm from other causes. Twenty cases he classes together as neuritic
neuralgic; in each case the cause for seeking advice was loss of writ-
g power or of some allied function, such as sewing, painting, or organ-
aying. A strain or injury, over-work, mental worry and general
tigue added to local fatigue were the causes. The symptoms in these
ses he thinks were dependent upon a mild form of neuritis. They
osely resembled those of true writer's cramp, and were separated from
e latter only because: 1. The symptoms involved a wider area. 2.
hey have been in some cases produced without excessive exercise of any
nction. 3. Nerve tenderness or neuralgia was a prominent symptom.

Of true writer's cramp *Dr. Poore* gives abstracts of thirty-two cases,
ith report of the muscles most affected and their reaction to electricity.
e says that in every case of impaired writing-power there was evidence,
ore or less marked, of derangement of one or more of the muscles used
r writing. One of the earlier symptoms of muscular derangement is
at the patient has altered his method of holding the pen. Inquiry as
the ability to perform other acts besides writing will also show the
esence of muscular derangement; but in this great caution is necessary,
a muscle may be incapable of prolonged action, yet be able to perform
rapid act with great force. The occurrence of associated or consenta-
ous movements, such as movements of the left fingers while the right
nd is writing, may always be taken as evidence of muscular weakness.
hey only occur during the performance of that act which the patient

finds difficult. Depressed or exalted irritability is certainly a sign of muscular derangement. Indirect evidence of muscular derangement may be afforded by derangement of the sensory branches of mixed nerves which supply the muscles implicated.

Dr. Poore considers that writer's cramp is a peripheral disease, not a disease of the co-ordinating centres. 1. Because he has never seen a case without evidence of peripheral change, and in most cases no evidence exists of any other change. 2. He considers writing is acquired by education and is not an essential attribute of man and he does not believe co-ordinating centres can be created by education. 3. The fact that the affection is of gradual growth and is never suddenly established militates against the idea of a controlling centre. He would couple writer's cramp with neuralgia, a disease all of whose phenomena are local, due to condiditions which may affect nerve-tracts before or after junction with their nerve-centres.

In treatment of true writer's cramp rest must be obtained for the hand and also for the patient's mind and body. Many times the patient is relieved by having the nature of his malady explained to him, understanding that it is purely local. He thinks there is no harm in permitting a moderate use of the left hand to relieve the right. One of the most useful aids to recovery in advanced cases is rhythmical exercise of the affected muscles, but not to fatigue; friction with slightly stimulating liniment. The passage of the galvanic current through the muscles during rhythmical exercise is strongly advised. A warning is given against the continuous use of mechanical contrivances.

Dr. Beard concludes from his analysis of one hundred and twenty-five cases occurring in his own practice and in that of correspondents: 1. What is called the cramp is but one of a large number of the symptoms of this disease, and no two cases are precisely alike.

2. Also in other forms of professional cramp, as that of telegraphers, musicians, sewing women, painters, etc., the cramp is but one of a number of symptoms, and by no means always the most important symptom; and as in writer's cramp, there is frequently no cramp at all from the beginning to the end of the disease.

3. This disease is primarily a peripheral and local disease of the nerves and muscles; secondarily and rarely it becomes central and general, or it may result from various central lesions; and it may affect any point between the extreme periphery and the centre.

4. This disease occurs mostly in those who are of a strong, frequently of very strong constitutions, and is quite rare in the nervous and delicate; and when it does occur in those who are nervous, is easier relieved and cured than when it occurs in the strong.

5. This disease is far less likely to occur in those who do original work, as authors, journalists, composers, than in those who do routine work, as clerks, bookkeepers, copyists, agents, and so forth.

6. This disease, like all nervous diseases in this country, diminishes in frequency as we go south.

7. Writer's cramp is no longer an incurable disease.

8. The treatment of writer's cramp and affections allied to it consists: 1. In the use of electricity locally applied. Both galvanic and faradic currents may be used—preferably the former. 2. Hypodermic injections of atropine, strychnine, duboisia, Fowler's solution and other tonics, narcotics and sedatives. 3. The internal use of calabar bean, ergotine, iodoform, and in some cases of nerve-food, as oil and fats. 4. Massage. 5. Dry heat and dry cold. 6. Actual cautery and very small blisters to the upper portion of the spine or along the course of the affected nerves and muscles.

He then mentions various "hygienic devices" as holders, type-writers, writing with the left hand, etc.

Gowers, differing with *Poore* and *Beard*, thinks the disease is central; he believes the danger from using the left hand is exaggerated; reports a case where the patient, after using the left hand a year and resting right hand, was able gradually to return to the use of the right hand.

Carl Hertzka found gelsemium sempervirens useful in one case of piano-player's cramp, in which there were vague pains and weakness in both arms, which had prevented playing for two years. The patient for three weeks took tincture of gelsemium, eight drops three times a day. The symptoms disappeared without any unpleasant results.

Bianchi used hypodermic injections of nitrate of strychnia, using a solution such that one gramme contained five milligrammes of the nitrate of strychnia. He began by using one milligramme and increased the dose to three and one-half milligrammes every other day. The injections were made in the forearm. He concludes that: 1. The morbid condition or process differs as to its seat in different cases. 2. The difference in the disturbing process of the disease, in its seat, and in the mechanism by which the malady is determined, is perfectly in harmony with the difference of the clinical form. 3. The prognosis may be considered less grave than most pathologists believe. 4. The treatment, electric or internal, in order that it may obtain a greater average of recoveries must be conformable to the clinical character of the disease which differs in various cases, and which must indicate whether the ascending or the descending current may have a better effect on the spinal cord and on the nerves; when simultaneous faradizations of the muscles and of the skin may be applied with success; when galvanization of the brain or of the sympathetic nerves in the neck is useful and when the hypodermic injection of strychnia, exclusively or associated with established methods of electrization, may be of more utility.

PARALYSIS.

BIBLIOGRAPHY.

Erb: Ueber rheumatische Facialislähmung. Deut. Arch. f. kl. Med., XV., 1874, p. 6.—G. V. Poore: A Case of Paralysis of the Serratus Magnus. Lancet, Feb. 20th, 1875, p. 271.—E. Owen and S. J. Knott: Two Cases of Paralysis of the Serratus Magnus. Brit. Med. Journ., Mar. 6th, 1875.—R. Macdonnell: Case of Double Facial Paralysis with Loss of Taste in the fore part of Tongue. Brit. Med. Journ., May 29th, 1875, p. 726.—Onimus: On the Differences of Action of Induced and Continuous Constant Currents on the Muscular System, from a Clinical Point of View. Practitioner, June, 1875, p. 401.—Thomas Buzzard: Details of Electrical Examination and Treatment in a case of Peripheral Paralysis of the Facial and Oculo-Motor Nerves. Lancet, Oct. 2d, 1875, p. 484.—W. Erb: Eigenthüml. Localisat. von Lähm. im Plex. Brachialis. Verhandl. d. Heidelb. naturh.-med. Vereins, N. Ser. 1, 2, 1875.—Richelot: Distribution des Nerfs Collatéraux des Doigts, etc. Arch. de Phys., 1875, p. 177.—Alfred v. Gräfe: Die Lähmungen der Augenmuskeln. Handb. d. gesammt. Augenheilkunde v. A. v. Gräfe und Th. Saemisch, Bd. VI., I., p. 13, 1875.—M. Bernhardt: Ueber den Bereich der Sensibilitätsstörung an Hand und Fingern bei Lähmung des Nv. medianus. Arch. f. Psych., V., 1875, p. 555.—A. Cossy and J. Dejerine: Recherches sur la Dégénérescence de Nerfs séparés de leurs Centres trophiques. Arch. de Physiol., 1875, p. 567.—Senftleben: Ueber die Ursachen und das Wesen der nach der Durchschneidung des Trigeminus auftretenden Hornhautaffection. Virch. Arch., LXV., 1875, p. 69; see also same journal, LXXII., 1878, p. 279.—Fr. Bärwinkel: Kritisches zur Differential-Diagnose der Faciallähmungen und Klinisches über die Chorda Tympani. Deut. Arch. f. kl. Med., XVII., 1875, p. 122. H. Quincke: Vagus Reizung bei Menschen. Berlin. kl. Wochen., 1875, pp. 189, 203.—Erb: Ein Fall von Bleilähmung. Arch. f. Psych. u. Nervenkr., V., 1875, p. 445.—E. Remak: Zur Pathol. d. Bleilähmung. Arch. f. Psych. u. Nervenk., VI., 1875, p. 1.—S. O. Habershon: On the Pathology of the Pneumogastric Nerve. Brit. Med. Jour., April 15th, 1876, p. 465.—C. H. Jones: Sensory and Motor Paresis of Left Arm Removed by Blistering a Tender Spot. Med. Times and Gaz., Apr. 22d, 1876, p. 437.—Edmund Owen: Three Cases of Injury to Nerves (Trophic Changes). Brit. Med. Jour., Dec. 16th, 1876, p. 787.—S. G. Webber: Pain as a Symptom in Facial Paralysis and its Cause. Boston Med. and Surg. Jour., Dec. 28th, 1876.—C. Eisenlohr: Zur Pathologie d. Typhuslähmung. Arch. f. Psych. u. Nervenkr., VI., 1876, p. 543.—Fr. Fischer: Zur Lehre von den Lähmungen d. N. Radial. Arch. f. kl. Med., XVII., 1876, p. 392. Schnitzer: Ueber traumat. Lähmung d. Armnerven, speciell des Medianus. Diss. Berlin, 1876.—Bernhardt: Seltener Verlauf einer peripher. traumat. Lähmung. Arch. f. kl. Med., XVII., 1876, p. 307; Ueber Lähmung der Gesichts- u. Hörnerven. Arch. f. Psych. u. Nerv., VI., 1876, p. 549.—P. Reclus and H. Fourestié: Section accidentelle de l'artère cubitale, du Nerf médian et du Nerf cubital, étude de la Sensibilité de la Main. L'Union Méd., 9, 1876.—O. Berger: Zur Pathologie der rheumatischen Facialislähmung. Deut. med. Wochensch., 49, 1876 (Centralblatt f. d. med. Wissen., 1877, No. 15, p. 267).—Onimus: De la Contraction à la Suite des Paralysies pheriphériques en général. L'Union méd., 1876, No. 146 (Centralbl. f. d. med. Wissen., 1877, No. 15, p. 267).—B. Frankel: Lähmung des rechten Nervus accessorius. All. med. Cent. Zeit., No. 4, 1876, 47.

—H. DURET: Plaie contuse du Nerf médian; Troubles trophiques; Escharres aux Extrémités des Doigts. Gaz. méd. de Paris, I., 1876.—TYMACZKOWSKI: Ein Fall von Diphtheritis mit zugleich und successiv auftretender Lähmung; Heilung. Wien. med. Presse, 31, 1876, 1032.—ROSSBACH und QUILLHORST: Beiträge zur Physiologie des Vagus. Verhandl. d. physik. med. Gesell. in Würzburg, IX., 1876, p. 13-31.—M. BERNHARDT: Ueber einen bisher noch nicht beobachteten Verlauf einer peripherischen (traumatischen) Ulnarislähmung. Deut. Arch. f. k. Med., XVII., 1876, p. 307.—PIERRET: Lésions du Système Nerveux chez les Malades atteints de Paralysies diphthéritiques. Gaz. des Hôp., No. 2, 1877, p. 13. —GOWERS: Cases of Paralysis of the Musculo-spiral Nerve. Med. Times and Gaz., May 5th, 1877, p. 474.—BELLAMY: Injury to Ulnar Nerve. Paralysis. Recovery. Lancet, Oct. 20th, 1877, p. 570.—N. FEUER: Untersuchungen über die Ursache der Keratitis nach Trigeminus-Durchschneidung. Med. Jahrb. der k. k. Gesell. d. Aerzte, 1877, p. 197.—LUDWIG BRIEGER: Ein Fall von totaler doppelseitiger Stimmbandlähmung. Berl. kl. Wochenschr., 1877, p. 323 (Both recurrent Nerves were pressed upon by Enlarged Bronchial Glands).—ERNST REMAK: Zur Pathologie der Lähmungen des Plexus brachialis. Berl. kl. Wochens., No. 9, 1877, p. 116.—F. FALK: Zur experimentellen Pathologie des X. Gehirnnerven. Arch. f. exper. Pathol., VII., 1877, p. 183.—W. G. SMITH: Facial Paralysis; with Remarks on the Action of Induced Currents and of Interrupted Voltaic Currents. Dublin Jour. of med. Sci., Feb., 1878, p. 125.—DIEULAFOY: De la Paralysie du Nerf radial. Gaz. Hebd, 31 May, 1878, p. 341.—ONIMUS: Sur la Paralysie du Nerf radial. Gaz. Hebd., 21 June, 1878, p. 393.—PANAS: Sur une Cause peu connue de Paralysie du Nerf cubital. Arch. Gén. de Méd., Juil., 1878, p. 5.—ROBT. M. SMITH: Note on the Effects of Pressure on the Irritability of Nerve Trunks. Phila. Med. Times, July 6th, 1878, p. 457.—R. S. ARCHER: A case of Paralysis of the Trifacial Nerve; with Remarks. Brit. Med. Jour., Oct. 5th, 1878, p. 514.—M. BERNHARDT: Neuropathologische Beobachtungen. I. Periphere Lähmungen. Deut. Arch. f. kl. Med., XXII., 1878, p. 362.—W. KORYBATT-DASZKIEWIEZ: Ueber die Degeneration und Regeneration der markhaltigen Nerven nach traumatischen Läsionen. Diss., Strassburg, 1878.—W. SCHNITZER: Ueber Traumatische Lähmungen der Armnerven, speciell des Medianus. Diss., Berlin, 1876.—R. JACOBI: Ein Fall von schwerer traumatischer Paralyse des Nervus radialis und Nervus medianus. Diss., Marburg, 1877. [These last three found briefly summarized in Centralb. f. d. m. Wissen., 1878.]—T. WASYLEWSKI: Ueber Vagusreizung beim Menschen. Vierteljahrsch. f. d. prakt. Heilkunde, 1878, p. 69.—J. DEJERINE: Recherches sur les Lésions du Système Nerveux dans la Paralysie Diphthéritique. Arch. de Phys., X., 1878, p. 107.—M. RAYMOND: Monoplégie Brachiale. Gaz. Hebd., 31 Jan., 1879. p. 79.—L. MCLANE TIFFANY: Deformity of Shoulder Following Nerve Injury. Am. Jour. Med. Sci., July, 1879, p. 85.

Robert M. Smith has experimented upon the effect of pressure upon nerves in producing paralysis, somewhat as *Mitchell* has done formerly. *Smith* experimented upon frogs, using a column of mercury to obtain the pressure; when the column is from thirty-five to four hundred and fifty millimetres in height there is an increase of irritability of the muscular or peripheral end of the nerve, while the irritability of the spinal or central end as constantly decreases. Though these modifications bear a certain relation to the degree and duration of pressure, the variation is much less marked in the peripheral than in the central end of the nerve.

On removing the pressure, there is generally a diminution of irritability of the peripheral end and increase of irritability of the central end.

The reflex irritability of a nerve subjected to pressure, in the periphral end, is diminished during pressure and tends to regain the normal on its removal, while the irritability of the central end in some instances, when subjected to comparatively light pressure, is at first increased and then diminished with a tendency to rise upon removal of the pressure if its irritability has been reduced below the normal, while in other cases of more severe pressure the preliminary rise is wanting; also, if pressure has been continued too long, the rise subsequent to its removal may be wanting.

S. thinks an explanation of these phenomena may be found in a reversal of the natural electric current in the nerve.

Bernhardt gives statistics of the frequency with which various peripheral paralyses occurred in his experience during two and three-fourths years. He had 28 cases of lead paralysis, all males; 14 of these being painters, 4 file makers, 3 varnishers, some of these without colic, some had had colic two to four times. Generally the extensor muscles of the hand or fingers were affected alone, when others were affected the deltoid was most frequently the muscle attacked, next those of the thenar eminence. The left hand was affected alone in the case of a left-handed person. There were 19 cases of radial paralysis; 14 where the ulnar nerve was affected, nearly all traumatic, 13 of the median nerve, 5 of the axillary, of the musculo-cutaneus alone none, in 2 cases all the nerves of the arm were affected, and in 3 the serratus magnus; 1 of these was from a chill, 1 from pressure, 1 from tearing. In 2 cases the nerves of the lower extremities were paralyzed, both were cases of neuritis.

W. Korybatt-Daszkiewiez has studied anew the degeneration and regeneration of the medullary nerve-fibres after injuries. There is little that is new in his dissertation.

Cosse and *Dejerine*, from their investigations in regard to the degeneration of nerves separated from their trophic centres, conclude that the loss of motricity in the peripheral end of a divided nerve, which is seen on the third day, results from an alteration in the molecular condition of the axis cylinder—an alteration which renders it more fragile and perhaps ends in producing a solution of continuity, but which in every case renders it incapable of transmitting excitations, whatever may be their nature. II. This alteration is primitive; the changes of the nuclei and protoplasm are secondary. III. There is a rather striking analogy between the loss of the properties of the axis the third day and the marked diminution of contractility seen at the same period in the muscles whose nerves have been cut; these muscles then present no appreciable change in their primitive fibres. IV. The central nervous system may be considered as exercising an exciting influence on the nutrition of the nerve-tubes; removed from the influence of these centres the nerve-tubes rapidly lose their structure and their physiological properties. V. All the medullary tubes are subject to the influence of these trophic centres. This influence is not as well demonstrated for the fibres without myelin or Remak's fibres.

In the treatment of paralyses, *John E. Morgan* advises the application of electricity directly to the nerves and muscles by means of acupuncture. Finding electricity as ordinarily used of no avail, he passed needles into the limbs in two cases of total paralysis with wasting, and then found that the muscles responded feebly to the galvanic current. Forty or fifty cells were used without trouble two or three times a week for months. (He does not mention the battery used.) The result was very great improvement. This treatment is adapted to cases where there is motor and sensory paralysis with changes of electrical irritability of muscles or nerves, whether quantitative or qualitative.

Senftleben experimented upon the *trigeminal* in regard to the occurrence of keratitis. His conclusions are:

1. The affection of the cornea is independent of the influence of trophic nerve-fibres; there are probably none of these in the trigeminus.

2. The primary affection of the cornea after division of the trigeminus is probably a necrosis, caused by repeated coarse injuries which the eye receives in consequence of its anæsthesia.

3. The circumscribed necrosis of the cornea acts as an inflammatory irritation and calls forth a secondary inflammation of the cornea which advances towards the periphery.

4. The simultaneous extirpation of the upper sympathetic ganglion is of no influence upon the origin and course of the affection of the cornea occurring after division of the trigeminus.

Again in 1878 he repeats this opinion that the keratitis is of traumatic origin.

Feuer concludes from his experiments that:

1. Division of the trigeminus does not interfere directly with the nutrition of the cornea, nor does it cause immediate inflammation of the same, but it puts the eye in a condition of diminished resistance to external influences.

2. The keratitis has its ground exclusively in the cessation of winking, the uncovered cornea dries, necrosis of tissue follows, this necrotic part acts as an irritation, therefore a reactive inflammation causes necrosis of neighboring parts.

3. Diminished secretion of tears hastens this process.

R. S. Archer reports a rare case in which both divisions, sensory and motor, of the fifth were paralyzed together; vision, hearing, taste, smell, were all affected. Taste was blunted if not lost in the corresponding side of the tongue anteriorly. There was a profuse discharge of mucus from the nostril on the paralyzed side.

Onimus (*Practitioner*, Jun., 1875, p. 413) thinks that *facial* paralysis due to cold depends upon an affection of the terminal branches of the nerve rather than upon an affection of its trunk.

Bärwinkel, in a case of severe facial paralysis, differently from *Erb*, found the posterior auricular nerve was not entirely paralyzed. He concludes from this that the pressure was not equal on all the fibres, and hence it may be wrong to conclude that in light cases the nerve is affected externally to the canal. The amount of compression may vary within the canal; a plastic exudation giving rise to a severe form of paralysis, a serous exudation to a light form.

In regard to the chorda tympani, he reports a case of lesion of the facial within the meatus auditorius, the auditory and all the branches of the facial being affected, the fifth and the chorda escaping. Hence the chorda could not have followed the facial to the brain. He thinks it runs in the facial only as far as the ganglion geniculatum, and then passes into the fifth by the smaller superior petrosal nerve. He mentions cases in support of this view.

In regard to the secondary contraction after facial paralysis, *Onimus* states that it appears at the time of commencing improvement, while the muscles are still in a state of paresis and the nervous influence is still imperfect. The muscles are in a state of inflammation caused by the paralysis. He does not allow that there is increased irritability of the centre. As yet experiments have not proved any centripetal influence of a clearly motor centre.

Webber suggests that the pain which frequently attends facial paralysis or even precedes it is due to an affection of the auricular branch of the vagus which crosses the seventh within the petrous bone, gives branches to the seventh and is a nerve of sensation.

VAGUS.—Several papers have been published in regard to experimental physiology of the vagus. The titles only of the most important of these are given.

Löwit reports a case with enormous increase of the heart's action which he refers to a unilateral paralysis of the vagus; he further considers the subject of paralysis of this nerve in its effects upon the lungs and heart.

In regard to paralysis of other nerves there is comparatively little that is new, though much has been written.

Poore, in discussing a case of paralysis of the serratus magnus, differs with *Duchenne*, who states that when the arm is held in front if this muscle is paralyzed, there is rotation of the scapula, also that the arm cannot be raised above the horizontal. In *Poore's* patient this was the only muscle paralyzed, and the arm could be raised higher.

Panas reports four interesting cases of paralysis of the ulnar nerve from unusual causes. In the first there was development of an abnormal sesamoid bone in the internal lateral ligament of the elbow. Chronic neuritis of the ulnar was seemingly caused by repeated bruising of the nerve against this body. In the second, twelve and a half years after fracture of elbow, the groove in which it runs was filled up, the nerve became subcutaneous, and so more exposed. In the third case, three years' continued pressure on the elbow was found to have caused enlargement of the nerve. The fourth case was one of deformity by arthritis of the humeral trochlea.

In paralysis after diphtheria there have been several autopsies. *Dejerine* in five cases found constant changes in the cord and anterior roots, there being parenchymatous and interstitial inflammation of the gray substance in the cord. The lesion of the roots he thinks is secondary.

Pierret found the dura mater thickened, adherent, rough with a deposit of neo-membrane similar to that on the mucous surfaces.

NEURITIS.

BIBLIOGRAPHY.

R. Klemm: Ueber Neuritis migrans. Inaug. Dissert., Strassburg, 1874.—Fr. Bärwinkel: Die Bedeutung der centripetalen Irradiation bei schmerzhaften Affectionen der Nervenstämme. Deut. Arch. f. kl. Med., XVI., 1875, p. 186.—Franz Fischer: Zwei Fälle von Neuritis. Berlin. kl. Wochen., 32, 33, 1875.—W. Niedick: Ueber Neuritis migrans und ihre Folgezustände. Arch. f. exper. Pathol., VII., 1876, p. 205.—H. Nothnagel: Ueber Neuritis in diagnostischer und pathologischer Beziehung. Volkmann's Saml. kl. Vorträge, No. 103, 1876.—O. Rosenbach: Experimentelle Untersuchungen über Neuritis. Arch. f. exper. Pathologie, VIII., 1877, p. 223.—H. Eichorst: Neuritis acuta progressiva. Virch. Arch., LXIX, 1877, p. 265.—Ch. Fernet: Pneumonie aigue et névrite du pneumogastrique; pathogénie de la pneumonie. Arch. gén. de Méd., Juil., 1878, p. 90.—Bailly and Onimus: Lesions graves du plexus brachial produites par les manœuvres de dégagement du tronc après l'expulsion de la tête. Modifications de la contractilité electro-musculaire, importance de ces modifications pour le diagnostic et le prognostic. Gaz. Hebdom., 1878, p. 333.—Thomas Buzzard: Clinical Lecture on Neuritis, syphilitic and rheumatic. Lancet, Mar. 1st, 1879, p. 289.—J. Althaus: Contributions to the Pathology of peripheral Nerve Diseases. Am. Jour. Med. Sci., Apr., 1879, p. 363.—Wm. S. Savory: Cases of perforating Ulcer of the Foot with Remarks and an Appendix on the Literature of the Subject. Med. Chir. Trans., 1879, p. 373.—Hector Treub: Ueber Reflexparalyse und Neuritis migrans. Arch. f. exper. Path. u. Pharmak., X., 1879, p. 398.—C. Boeck: Et Tilfælde af Neuritis traumatica ascendens. Norsk Mag. f. Lägeridensk. 3. R. IX., 1879, p. 105.

Neuritis was distinguished from neuralgia by *Bärwinkel* by the direction in which pain radiates. Where the pain radiates centripetally, he says there is a slight inflammation of the neurilemma, a neuritis. Where this centripetal irradiation is wanting, it is a case of neuralgia. In a case of traumatic ascending neuritis, *Boeck* found that pressure over the nerve caused pain shooting towards the centre, according to *Bärwinkel's* statement. The spontaneous pain which *Nothnagel* claims as present was not felt.

An interesting case of *perforating ulcer* of the foot is recorded by *Savory*. The nerves were found diseased, the epineurium thickened, perineurium very little changed, the endoneurium thickened, the nerve-fibres were much atrophied and diminished in number.

Fernet claims that acute pneumonia, called fibrinous, is a herpes of the lung depending upon a neuritis of the pneumogastric nerve.

Neuritis migrans, or *progressiva*, has attracted considerable attention, and several new experiments have been recorded. *Treub* found that in six cases where he had been able to excite interstitial neuritis, it was transmitted by continuity, the infiltration never jumped over one part of the nerve to attack it anew; but the inflammation may attain a higher degree in certain points of predilection, where blood-vessels are more numerous. He thinks many cases of so-called reflex paralysis are really cases of neuritis extending by continuity.

Rosenbach found considerable difficulty in exciting neuritis, irritating injections producing only perineuritis, limited to the vicinity of the injection; threads drawn through the nerve excited neuritis, but there was no transplanting of the inflammation to neighboring parts of the nerve, nor to the spinal cord, nor its meninges in twenty-one cases. This does not prove, he adds, that clinically there may not be a neuritis migrans, though he considers "neuritis disseminata" as a better name. He operated on rabbits.

Klemm, who also operated on rabbits, came to a different conclusion. He injected a few drops of a solution of arseniate of potash. The resulting inflammation always jumped to spots where many arterial vessels branched. Even the dura mater spinalis was affected. Once the disease passed from the sciatic to the plexus brachialis. The cerebral dura mater was twice affected. The nerve of the opposite side was also affected, though the dura mater of the cord was not.

Niedick experimented upon rabbits, and found that the neuritis did not extend continuously, but by spots jumping over a stretch of healthy tissue. He found also centres of softening in the cord.

Eichorst reports a case in which there was slight affection of the perineurium, blood-vessels dilated and filled with blood, lymphoid cells next the vessels, blood extravasation in the endoneurium, nerve-fibres degenerated. One nerve after another was affected, an attack of fever attending the attack of each nerve; the superficial perineal nerve was first attacked, next the deep perineal, three days later the posterior tibial; later other nerves were affected. Sensibility was first lost, then the motility. The electrical reaction was lost soon after the paralysis appeared.

NEUROMATA AND NEUROPLASTIC FORMATIONS.

BIBLIOGRAPHY.

C. DE MORGAN: Case of Multiple Neuroma of the Forearm. Trans. of the Path. Soc., XXVI., 1875, p. 2.—A. V. WINIWARTER: Plexiformes Fibroneurom der Armnerven mit circumscripter Hauthypertrophie und Sarcomentwickelung. Ein Beitrag zur Geschwulstlehre. Arch. f. kl. Chirurg., XIX., 1876.—VERSTRAETEN: Lipôme du Nerf recurrent gauche. La Presse méd. Belge, No. 42, 1876.—TRELAT: Myxome du Nerf sciatique. Bul. de la Soc. de Chir., Séance du 17 du Nov., 1875. Centralbl. f. Chirurg., 1876, p. 464.—M. COLOMIATTI: Sur la Diffu-

sion du Cancer le Long des Nerfs. Arch. de Phys., 1877, p. 663.—Isidor Soyka: Ueber den Bau und die Heilung der multiplen Neurome. Vierteljahrs. f. d. prakt. Heilk., 1877, p. 1.—C. Gerhardt: Zur Diagnostik multipler Neurombildung. Deut. Arch. f. kl. Med., XXI., 1878, p. 268.—Duplay: Névrôme plexiforme Amyelinique de l'Avant-bras. Arch. Gén. de Méd., Avril, 1878.—A. Takacs: Ueber multiple Neurome. Virch. Arch., LXXV., 1879, p. 431.—G. Salomon: Multiple Neurome bei einem schwachsinnigen Individuum. Fortschreiten der Neurombildung während der Dauer der Beobachtung. Extirpation und mikroscopische Untersuchung eines Neuroms. Centralbl. f. d. med. Wissen., 31, 1879, p. 560.—Wm. M. Findley: Neuroma Involving the Ulnar Nerve; Excision with Entire Relief from Pain. Am. Jour. of Med. Sci., Jan., 1880.

Takacs reports a case of multiple neuromata; the tumors were filled with round cavities or canals crossed by fibres and filled with blood-clots; one, the size of a hazel-nut, was within the vertebral canal, inside the sac of the dura mater. The nerves were in part lost in the tumors. As the nerve-fibres approached the tumor they spread out, the nuclei became more numerous around them; the medullary sheath could not be recognized; the axis cylinders could be followed into the tumor, but were lost there, and within the tumor only a row of long nuclei suggested the nerve-structure.

It was evident that the new growth sprang from the interior of one or more of the primitive bundles of a nerve-trunk, from the interstitial tissue of the nerve-fibres, the endoneurium, and not from the perineurium, and the nerve-fibres of the primitive bundles were not simply pressed apart by the new growth and shoved to the periphery, but were frequently attacked concentrically.

To settle the diagnosis as to whether a tumor is a neuroma, *Gerhardt* recommends to introduce a needle into the tumor; if it involves a mixed or motor nerve, a weak current, which would not otherwise excite contraction, will cause the muscles to act. If the nerve is sensitive alone, only pain will be felt, perhaps with reflex contraction. In the case reported, when the needle entered the tumor, five cells produced contraction; if it only penetrated near the tumor five cells had no effect; while on the skin twenty-five cells were required to produce contraction.

Findley operated to remove a neuroma with as little injury as possible to the nerve: he passed a rubber ligature around the tumor, notching it first, so that the ligature might not slip; the nerve-fibres were left as intact as possible. The pain was relieved, and there was only a slight loss of motion.

DISEASES OF THE BRAIN.

DISEASES OF THE MEDULLA OBLONGATA.

BY

L. PUTZEL, M.D.,

OF NEW YORK,

PHYSICIAN TO THE CLINIC FOR NERVOUS DISEASES, BELLEVUE HOSPITAL OUT-DOOR DEPARTMENT; VISITING
PHYSICIAN FOR NERVOUS DISEASES, RANDALL'S ISLAND HOSPITAL; PATHOLOGIST TO THE
LUNATIC ASYLUM, B. I.; CURATOR TO CHARITY HOSPITAL, ETC.

DISEASES OF THE BRAIN.*

CEREBRAL ANÆMIA.

BIBLIOGRAPHY.

DAUTHEVILLE: Considérations sur l'ischémie et l'hypérémie cérébrales. Paris, 1879.—PASTOL: Etude clinique sur l'ischémie cérébrale localisée et son diagnostic differentiel. Thèse, Paris, 1879.—EULENBURG: Lehrbuch der Nerven-Krankheiten, Bd. II. Berlin, 1878.—BALL, BENJAMIN: Certain Cases of functional Ischæmia of the Brain. British Med. Journ., Oct. 30th, 1880.

Prof. *Benjamin Ball* gives the history of three cases which he has described as functional ischæmia of the brain. In the first case, "a young man, æt. 26 years, married, of good character and sober habits, gives way to a most violent fit of anger, and is suddenly struck deaf and dumb, with hemianæsthesia, and a slight motor paralysis on the left side. Speech is restored in the space of eighteen hours; the other symptoms persist during twenty-two days, and suddenly disappear after a few galvanic applications, limited to the posterior part of the left forearm. The patient's health was previously good; but the year before, in the month of May, he had been suddenly struck blind on the left side, and only recovered his visual powers after the lapse of a month."

CASE II.—A cab-driver, æt. 33 years, married; leads a sober life, and general health excellent up to present attack. During the month of December, 1879 (the winter was extremely cold), the patient had an attack of acute articular rheumatism. After his recovery, he returned to work. On February 24th, he had a severe attack of vertigo, and, on the next day, manifested a peculiar state of mental obstruction, which persisted on his admission to the hospital (March 1st, 1880). His face expressed vacant, stupid astonishment; he was unable, without assistance, to eat, drink, or attend to the calls of nature. His wife took care of him as of a small child; he was evidently distressed when she left the room, but could not call her back. The patient could not utter spontaneously a single word, but repeated any question which was asked him. He evidently did not understand the words, but repeated them automatically. There was loss of power on the right side of the body, with complete abolition of sensation. After the lapse of two weeks, he began to improve in all respects, and when he was discharged (April 30th), was in a satisfactory condition; his mental faculties had nearly recovered their former vigor; he had again learned how to speak and write correctly, but his memory still remained imperfect.

---

* In view of the fact that the articles on the Brain and Medulla Oblongata were placed in my hands at a very late day, the indulgence of the reader is desired if they should appear incomplete in any respect.—L. P.

Case III.—A shoemaker, æt. 45 years, entered the Hospital St. Antoine, on April 1st, 1877. His wife stated that, eight days before, while apparently in good health, he quarrelled violently with one of his apprentices, and was then suddenly struck dumb; no other morbid symptoms appeared. The tongue was easily protruded, and could be moved without difficulty from one side to the other. But when the patient attempted to speak, the middle part of the tongue was lifted up in the form of a convex dome, and struck the roof of the palate, thus obstructing the passage of sounds; he uttered with great difficulty something between a grunt and a growl. When the tongue was at rest, it was as soft and flexible as in the normal state. The patient was able to express himself well in writing; no other symptoms could be discovered. It was afterwards learned from the man's wife that his powers of speech were restored as suddenly as they were suppressed.

According to *Ball*, these cases must be attributed to spasmodic ischæmia of various portions of the brain (the parts affected being different in each of the three cases), and he considers himself justified in drawing the following conclusions:

1. Spasmodic contraction of the brain-vessels may be produced by moral impressions—fear, anger, or grief; and also by the prolonged action of severe cold.

2. All the symptoms of organic injury of the brain may be created by functional ischæmia.

3. Mental disturbance of a peculiar kind, and especially lowering of intellectual power, as apart from positive insanity, may be the result of this process.

4. Spasmodic contraction of the brain-vessels, when once induced, may persist for a considerable length of time without producing structural changes in the nervous centres.

5. This morbid condition may, in certain cases, suddenly disappear; while it is not unreasonable to suppose that the converse may be equally true, and that the symptoms may culminate in rapid or even sudden death.

CEREBRAL HYPERÆMIA.

BIBLIOGRAPHY.

Arndt: Zur Pathologie des Hitzschlages. Virch. Archiv, Bd. 64, p. 15-30. —Koester: Zur Pathologie des Hitzschlages. Berl. klin. Wochenschr., 24, 1875,

p. 465.—SIEDAMGROTZCKY: Zwei Faelle von Hitzschlag. Berl. klin. Wochenschr., July 17th, 1876.—HUGHES: Reflex Cerebral Hyperæmia. St. Louis Med. and Surg. Journ., 1879, p. 79–82.—COUPLAND: Diffuse Punctiform Hemorrhages in the Substance of the Cerebral Hemispheres in an Instance of Extreme Congestion of the Brain, due to Obstructed Pulmonary Circulation. Trans. London Path. Soc., 1879, XXX., p. 189–194.—FAYRER: Sunstroke and some of its Sequelæ. Brain, 1879–80, II., p. 297–316.—CURRAN: Sunstroke as I have seen it in India. Med. Press and Circular, 1880, p. 408 et seq.—AGNEW: The Value of the Ophthalmoscope as a Means of Determining the Presence or Absence of Cerebral Congestion. Med. Gaz., 1880, VII., p. 150.

Some neurologists have advanced the opinion that the presence of congestion of the brain may be determined by the use of the ophthalmoscope. We are glad to quote, in this connection, the following remarks made by the distinguished ophthalmologist, *C. R. Agnew:* "We would premise by saying that a reliable ophthalmoscopic examination is a most difficult thing to make. Unless the observer is able to use the refracting ophthalmoscope with great skill and fastidiousness, he is continually liable to mistake the distortion of objects in the patient's fundus oculi for pathological changes, and having set out with some prejudice in favor of cerebral congestion, to mistake such phenomena for certain indications of the same. That we may, by looking into the patient's eye with the ophthalmoscope, tell whether he has congestion of the brain or not, I do not believe. In the large number of cases of jaded head that we have seen, we have tried to ascertain whether a congestion or anæmia of the brain is present, and come to the conclusion that congestion is rarely present, and that in the bare majority of cases the ophthalmoscopic signs, except those of errors of refraction, are absolutely negative."

INSOLATION.

It has been generally maintained hitherto that the primary lesion in insolation is active congestion of the brain, but this view has been vigorously attacked within recent years. *Arndt* has advanced directly opposite opinions. In three cases in which he was able to perform autopsies, this author found the following condition of the organs: the integument and the muscles were pale, but the larger vessels contained in these tissues were distended with blood. The brain and its membranes were also extremely pale and anæmic, though the larger vessels in these organs, especially the veins and sinuses, were distended with dark, non-coagulated blood; the brain was very moist, and the ventricles contained a considerable amount of clear serum; the pia mater could be very easily detached from the convolutions. *Arndt* believes that those writers who have described hyperæmia of the brain as occurring in insolation have been misled by the fact that, in cutting the organ, the blood escapes from the large vessels which are congested and then flows over the cut surface of the brain. The structure of the heart was pale, but the coronary arteries and their branches were enormously distended with blood. A similar condition was found in the liver, kidneys, and mucous membrane of the intestinal canal; these organs also appeared to be œdematous. These appearances are regarded by *Arndt*, not as the results of simple anæmia and œdema, but as cloudy swelling, or the first stage of parenchymatous inflammation. No microscopical examination of the affected organs was made.

The lesions in question are found in infectious diseases and in various affections which are associated with high temperature. In insolation, also, *Arndt* believes that they are due to the high temperature so characteristic of this disease, and that they constitute its most essential feature. Those cases which are chiefly marked by cerebral symptoms we must attribute to a developing parenchymatous encephalitis. The sequelæ of insolation, the great loss of the power of resistance, the changed psychical character, the increased disposition to mental disease are connected with the encephalitis in question, and we can more readily explain in this than in any other manner the fact that insolation is so often followed by severe nervous diseases and psychical disorders. "The nutrition of the brain has suffered so severely on account of the beginning parenchymatous inflammation that it becomes the locus minoris resistentiæ during the remainder of the patient's life."

CEREBRAL HEMORRHAGE.

BIBLIOGRAPHY.

Lépine: De la localisation dans les maladies cérébrales. Paris, 1875.—Leber: Die Krankheiten der Netzhaut und des Sehnerven. Graefe's u. Saemisch's Handbuch der Augenheilkunde, Bd. V., S. 940.—Flechsig: Die Leitungsbahnen im Gehirn u. Rückenmark. Leipzig, 1876.—Ollivier: Gaz. hébdom., 1875, 11-13, and Archiv d. Physiol., 1876. — Eichhorst: Ueber eine häufige Form von Zwangsbewegung bei Erkrankungen des Gehirns. Charité-Annalen, 1 Jahrb., 1876, p. 224.—Pitres: Recherches sur les lésions du centre ovale des hémisphères cérébraux étudiées au point de vue des localisations cérébrales. Paris, 1877.—Graefe and Saemisch: Handbuch d. Augenheilkunde, Bd. VIII., 1877.—Rosenbach, Ottomar: Ueber die Localisation acuter Lungenerkrankungen bei Hemiplegischen. Berl. klin. Wochenschr., Oct. 14th, 1878.—Eulenburg: Lehrbuch der Gehirnkrankheiten. Berlin, 1878. — Grasset: Des localisations dans les maladies cérébrales, 1878.—Charcot and Pitres: Contribution a l'étude des localisations dans l'écorce des hémisphères du cerveau. Rev. mensuelle de méd. et de chir., 1877, 1878, 1879.—Wilks: Lectures on Diseases of the Nervous System. London, 1878.—Bernhardt: Ueber den diagnostischen Werth der Symptome der Déviation conjugée und der abnormen Kopf- und Rumpfhaltung bei Hirnkrankheiten. Virch. Archiv, Bd. 69, 1877, p. 1-24.—Mackey: Case of Hemorrhage in one Lobe of the Cerebellum. Brit. Med. Journ., 1878, II., 950.—Fuerstner: Eine eigenthümliche Form von Sehstörung bei Paralytikern. Arch. f. Psych. u. Nervenkr., Bd. VIII., S. 162, and Bd. IX., S. 90.—Rosenthal: Diseases of the Nervous System; English by Putzel, 18[illegible]9.—Broadbent: Conjugate Deviation of the Head and Eyes as a Symptom in Cerebral Hemorrhage and other Affections. The Lancet, Dec. 18th, 1879, p. 861.—Da Costa: Death from Intracranial Hemorrhage; Autopsy. Med. and Surg. Rep., Phila., 1879, XLI., p. 7-9.—Bennet: Case of Cerebral Hemorrhage in a Child one year and ten months old. Brit. Med. Journ., 1879, II., 692.—Charcot: Dégénérations secondaires. Progrès méd., 1879, VII., p. 597-600.—Ferrier: The Localization of Cerebral Disease, 1879. —Nothnagel: Topische Diagnostik der Gehirnkrankheiten, 1879.—Landouzy: De la déviation conjugée des yeux et de la rotation de la tête par excitation ou paralysie des 6e et 11e paires; leur valeur en sémeiotique encéphalique; leur importance au point de vue anatomique et physiologique. Progrès méd., Sept. 6th, 1879 et seq.—Kahler and Pick: Beiträge zur Pathologie u. path. Anatomie des Centralnervensystems. Prager Vierteljahresschrift, 1879, p. 1-80.—Maragliano: Le localizzazioni motrice nella corteccia cerebrale, studiate specialmente del lato clinico. Riv. sperimentale di freniatre e di med. leg. Anno IV., fasc. 1. —Mills: Hemiplegia, Aphasia, and Unilateral Convulsions from Cortical Disease. Med. Bull., Phila., 1879, I., p. 13-15.—Boyer: Etudes topographiques sur les lésions corticales des hemisphères du cerveau. Versailles, 1879.—Foulis: A Case in which there was Destruction of the Third Left Frontal Convolution without Aphasia. Brit. Med. Jour., 1879, I., p. 383.—Barthelemy: Hémorrhagie cérébrale; déstruction lacunaire, ancienne, de toute l'écorce des circonvolutions temporales, n'ayant jamais donné lieu à aucune signe. Progrès méd., 1879, VII., 922.—Bucquoy: Hémorrhagie cérébrale ayant simulé cliniquement une méningite bulbaire. France méd., 1879, XXVI., p. 713.—Baker: Early Degeneration of Cerebral Arteries; Cerebral Hemorrhage. Ann. Anat. and Surg. Soc., Brooklyn, 1879, I., p. 41.—Sorel: Contribution a l'étude des localisations cérébrales. Rev. mensuelle, July 10th, 1880, p. 542.—Tripier, Raymond: De l'anæsthésie produite par des lésions des circonvolutions cérébrales. Rev. mensuelle, Jan. and Feb., 1880.—Peabody: A Case of Multiple Cerebral Hemorrhages with Dissecting Aneurism of Aorta. Med. Record, 1880, XVII., 433.—Hardy: Un cas d'hémorrhagie cérébrale limitée à la capsule externe. Gaz. méd. de Paris, 1880, 6 s., II., 322.—Pitres: Marche de la dégénération secondaire dans les lésions motrices corticales. Progrès méd., 1880, VII., p. 131.—Hughlings Jackson: Remarks on Diseases of the Cerebellum. Med. Pres. and Circ., Lond., 1880, XXIX., 448, 469.—Gallois: Hémorrhagie cérébelleuse. Prog. méd., 1880, VIII., 633.

Considerable attention has been paid to the variations in the occurrence of the different reflexes upon the paralyzed and non-paralyzed halves of the body in cerebral hemorrhage. A few years ago, *Jastrowitz* showed that the reflex which follows stroking, striking, etc., the inner side of the thigh in its upper third, viz., contraction of the cremaster muscle and consequent ascent of the testicle on the same side, remains absent, as a rule, in hemiplegics on the paralyzed side.

Patellar tendon-reflex and ankle-clonus are usually present, however, and may even be exaggerated upon the affected side. This occurs most frequently when there is a certain amount of contracture present in the paralyzed muscles, with secondary degeneration of the spinal cord.

Rosenbach also found an interference with the reflex movements obtained upon stroking the abdominal integument in hemiplegics. During health such a manipulation is always followed by a usually quite vigorous contraction of the muscles of the abdomen ; upon the paralyzed side of hemiplegics, however, the reflex muscular contractions are not so vigorous, or are entirely absent. The involuntary muscular fibres on the paralyzed half of the body also take part in the general disturbance of reflex action. Thus, diminished contraction of the smooth muscular fibres of the nipple ensues upon rubbing or other irritation of this part. Furthermore, the so-called goose-skin can only be produced upon the non-paralyzed side of the body. Finally, the reflexes following irritation of the conjunctiva, the nasal mucous membrane, and the external auditory meatus are interfered with and not so rapid as on the sound side.

A very interesting series of phenomena has been explained by *Rosenbach* by this diminution of reflex excitability of the paralyzed half of the body. He found that acute pulmonary diseases, such as croupous pneumonia, pleurisy, broncho-pneumonia, which develop in hemiplegics, always affect the paralyzed side. This opinion is based on the study of eleven cases, in which the clinical history was of such a nature that it left no doubt with regard to the diagnosis, and the majority of which were demonstrated upon autopsy. They included four cases of croupous pneumonia of the right and left lungs, the upper and lower lobes, four cases of pleurisy with effusion (right and left sides), and three cases of broncho-pneumonia ("Verschluckungs" pneumonia). The lung appears to retain the predisposition to disease for a long time, as the affected side was the site of the pulmonary affection, even when the hemiplegia was of long standing or had almost entirely disappeared.

The diminution of reflex excitability referred to above has been called into requisition by *Rosenbach* in order to explain this peculiar localization of the pulmonary lesions in the broncho-pneumonic affection. It may be assumed that the reflex excitability of the bronchial mucous membrane, like that of the conjunctivæ, etc., is also diminished. Foreign bodies, therefore, which enter the affected lung, will not give rise to violent cough, they remain in the bronchial tubes, and thus produce spots of broncho-pneumonia of variable extent. The development of this lesion is also aided by the diminution in the movements of the trunk on the paralyzed side, which, although denied by *Broadbent*, has been observed by other writers. The occurrence of croupous pneumonia on the hemiplegic side is difficult of explanation.

Prevost, who first called attention to conjugate deviation of the head and eyes, had stated that, whenever the lesion was situated in the hemispheres, the eyes were turned away from the paralyzed side, *i. e.*, towards the side of the lesion; when the latter was situated in the mesocephalon, the eyes may be turned towards the paralyzed side. These statements do not, however, hold good in all cases, so that *Bernhardt* has declared that this symptom cannot be employed as a diagnostic aid in determining the site of the lesion, in such cases in which the motor paralysis cannot be determined, on account of the deep coma into which the patient has fallen, or of the complete resolution of all the limbs.

Conjugate deviation is observed most frequently when the hemorrhage involves the corpus striatum or its immediate neighborhood, though it is occasionally observed when the lesion is situated in any other portion of the brain. Although the patient is profoundly comatose, and almost complete relaxation of the limbs is noticeable, distinct resistance is manifested when an attempt is made to restore the head to its natural position.

Broadbent explains this peculiar phenomenon by means of his theory of the association of nerve-nuclei in the cord, which also accounts for the almost complete immunity of the muscles of the trunk in hemiplegia, and has been generally accepted by pathologists. This author makes the following statements in this connection :

"If we examine into the difference between the paralyzed and non-paralyzed parts in hemiplegia, it will be found that those parts are paralyzed which are perfectly independent of corresponding parts on the other side, while those escape which always, or at least habitually, act together with the corresponding muscles on the opposite side, and cannot be thrown into action independently of them. Whenever the muscles on the two sides of the body are incapable of being moved independently of each other, there must be some nervous mechanism which associates their nerve-nuclei in the spinal cord. Thus, the nerve-nuclei for the muscles of the two halves of the trunk, instead of being perfectly independent and distinct, like those of the arms or legs, are connected by transverse bands, so that, to all intents and purposes, two nuclei are fused together, and there is a common nerve-nucleus for the two sides connected with both corpora striata. If one corpus striatum is damaged, the other transmits orders to this nucleus common to both sides of the body. This is shown both in paralytic and convulsive diseases. Conjugate deviation, then, arises from this bilateral association of nerve-nuclei. Every day experience teaches that, in the case of the eyes, there is not a simple transverse, but an oblique association; the nucleus of the third nerve, which is situated in the anterior portion of the pons, being associated with the nucleus of the sixth nerve, located in the lower part of the pons. In the rotation of the head, there is co-operation of the inferior oblique (atlo-axoid) muscle on the side towards which the head is turned, and of the sterno-cleido-mastoid on the opposite side. The nerve-supply of these muscles is an exact parallel to that of the third and sixth in the upper part of the medulla. The sterno-cleido-mastoid is supplied by the spinal branch of the spinal accessory nerve, which arises from the whole length of the cervical portion of the cord, while the inferior oblique muscle is supplied by the first cervical nerve, which arises from the upper part of the cord. The association of the nerve-nuclei in this case also is, therefore, long and oblique. It is during the time that the oblique communication is coming into operation after damage to the corpus striatum or hemispheres that the lateral deviation occurs."

CEREBRAL LOCALIZATION.

A large part of our knowledge with regard to the localization of cerebral diseases has been acquired within the last five years, and although this subject is still involved in great obscurity, we are possessed of exact data with regard to some of the subjects included in this category.

This is especially true concerning lesions of the motor centres situated in the cerebral convolutions.

If, in any case, motor disturbances can be attributed to a cortical disease, the ascending parietal and frontal convolutions and the paracentral lobules are always involved. In some cases, lesions of the parts referred to may present symptoms which are of an entirely similar nature to those produced by an affection of the corpus striatum, viz., ordinary hemiplegia, but, in the majority of instances, they result in monoplegia or in dissociated hemiplegia. The following forms of paralysis from lesions of the motor centres have been observed, viz.: Isolated ptosis, paralysis of the facial nerve, paralysis of the hypoglossus, paralysis of one limb (usually the upper), paralysis of both limbs (on the same side) without implication of any cerebral nerves, paralysis of an arm and one cerebral nerve (usually the facial), paralysis restricted chiefly to the distribution of one of the nerves of a limb.

The occurrence of monoplegia, when the other symptoms presented in the case indicate the cerebral nature of the paralysis, points, with great probability, to a cortical lesion. Writers are not in exact accord with regard to the localization of these various paralyses, but they are, in the main, agreed, and *Nothnagel*, therefore, considers himself warranted in the following conclusions: Paralysis of the facial and hypoglossal nerves is referable to a lesion of the lower third of both central gyri; paralysis of the upper limb to a lesion of the middle third, especially of the anterior gyrus; paralysis of the lower limbs to a lesion of the upper third of both gyri. The paracentral lobule appears to be connected with paralysis of the limbs. When the motor paralysis is associated with marked vaso-motor or sensory disturbances, it is almost certain that the lesion is not situated in the cortex. Localized (non-generalized) convulsive movements, which develop at the onset of an attack of cerebral hemorrhage, indicate probably, though not certainly, the occurrence of a cortical lesion. Partial convulsions, however, which develop at a later period, in the previously paralyzed parts, have only been observed hitherto in diseases of the cortex, and constitute, therefore, a very important diagnostic sign.

Although disturbances of cutaneous sensibility have been mentioned in a few cases of disease of the cerebral convolutions, they have all been of such a nature that no positive conclusions can be drawn with regard to the cortical site of this symptom. The same remarks will also hold good with regard to the muscular sense. The meagreness of our knowledge in these respects must be attributed, in great part, to the want of attention to these symptoms on the part of observers. In almost all the cases which presented an affection of cutaneous sensibility, the parietal lobes appear to have been involved, and it is, therefore, incumbent upon observers to pay special attention to this region in making autopsies upon cases of this character.

Very little is known concerning the disturbances of vision occurring in cortical lesions. They usually consist of hemiopia or hemianopsia. *Fuerstner* has, however, described a peculiar form of visual disturbance which is also due to a cortical affection. The following is a history of one of *Fuerstner's* cases:

A man, æt. 44 years, had been in good health previous to his present attack ot illness. On Feb. 5th, after an apoplectic seizure, he was found suffering from paresis of the right side of the face and the right arm, which disappeared after the lapse of two days; conjugate deviation of the head and eyes to the left was present and continued for two weeks; demented condition. After the conjugate deviation had disappeared, the following state of affairs was noticed: With the left eye, the patient observed all objects which were placed before him; with the right eye he could see nothing. There was no hemiopia or diplopia; ophthalmoscopic examination negative. The blindness of the right eye gradually improved until finally the difficulty of vision merely consisted in the inability of the patient to grasp with precision objects held before him, to count objects, etc.; in writing, he runs one letter into another, and often finds the pencil running upon the edge of the slate; appreciation of colors is unaffected. The improvement continued until Aug. 2d, when suddenly three apoplectiform attacks occurred, the convulsions being limited chiefly to the left side. On Aug. 4th, when the patient's mental condition was better, he stated that he was blind; paresis of the left arm and left side of the face. Aug. 6th, he could see, but less on the left side than on the right. Aug. 29th, increase of the paresis; sight considerably diminished; small objects could not be distinguished at all. Rapid deterioration of the mental faculties, and marked symptoms of progressive paralysis of the insane. Death.

No cases of hemiopia have been reported hitherto in which the cerebral lesion was confined strictly to the cortex; the corona radiata was involved, to a certain extent, in all cases. A common element, however, in those cases of visual disturbance associated with cortical lesions is the existence of some diseased process in the occipital lobes; in a few cases, the latter were the only parts involved. In addition, *Huguenin*, in examining the brain of a man who had been blind for fifty years on the left side, found the following appearances: In addition to changes in the optic nerve, there was atrophy of the pulvinar, the anterior and posterior corpus quadrigeminum, and the external geniculate body on the left side; furthermore, there was atrophy of the occipital convolutions on both sides, but more marked on the right; the cortex was narrower, the gyri thinner and the sulci broader. The weight of clinical evidence is decidedly in favor of an implication of the occipital convolutions, whenever visual disorders are due to a cortical lesion. But *Ferrier*, who bases his opinion chiefly on the results of his experimental investigations upon the brains of monkeys, believes that the angular gyrus presides over the sense of sight. In the absence of a sufficient number of convincing clinical cases, we must, therefore, regard this question as unsettled.

Apart from those cases in which the acoustic nerve is affected by lesions at the base of the brain, unilateral auditory disturbances are very rarely due to intra-cerebral diseased processes. No cases have been hitherto reported in which deafness has been due to affections of the cortex.

Wernicke has, however, called attention to a peculiar form of auditory disturbance, which he attributed to a lesion of the first temporal convolution on the left side. This view has been confirmed by *Kahler* and *Pick*, who have collated the entire literature of the subject. In this affection, the patient can hear sounds distinctly, but does not understand what is said to him, and therefore gives wrong answers to questions, etc. The following is an abstract of the history of a case reported by *Kahler* and *Pick:*

A woman, æt. 42 years, was previously in good health. In July, 1875, she began to suffer from headache; three months later she is said to have lost the power of speech, and then became deaf. In 1876, she manifested symptoms of insanity, ran into the street in her shirt, etc. The medical history taken in October, 1876, states that the patient did not answer questions which were put to her, but that she was continually muttering inarticulate sounds. In the lunatic asylum, she had one attack of unconsciousness, which was not associated with convulsions. She is apathetic, continually muttering to herself, even during meals. She reacts very little to external influences, and does not answer questions, but nods occasionally in response, showing that she hears what is said; the fact that she must be shown by the nurse how to do even the simplest things, proves that she does not understand what is said. The patient died in 1877.

Autopsy: The pia mater somewhat opaque and strongly congested, especially over the parietal lobe; the convolutions of the frontal lobes and the central convolutions are somewhat atrophied. The convolutions of the left temporal lobe are broader than usual, pressed together, and of very soft and gelatinous consistence; upon cutting into them, the tissues are found softened, and the boundary between the cortex and white matter is indistinct. The right temporal lobe is softened to a less extent. A transverse section shows that the disease is chiefly confined to the cortical substance. The brain presents no abnormal appearances in other respects.

THE CENTRUM OVALE.

Our knowledge of localization in this part of the cerebral structure may be said to date from the publication of *Pitres'* treatise in 1877. *Nothnagel* has suggested a somewhat different anatomical division of this part of the brain from that recommended by *Pitres*, and we shall adopt the former on account of its greater simplicity. Each hemisphere is supposed to be divided by vertical parallel planes. The first separates the posterior central convolution from the parietal lobe, the second passes through the parieto-occipital fissure (on the median aspect of the brain), and then outwards in such a manner as to separate the parietal from the occipital lobe; the third passes through the fissure of Rolando; the fourth separates the anterior central convolution from the frontal lobe; the fifth begins immediately in front of the genu of the corpus callosum and then runs outwards parallel to the others; the sixth starts at the base of the brain from the origin of the Sylvian fissure. The term "occipital part" is applied to that portion of the brain behind the second plane; "parietal part" to that portion between the first and second planes; "posterior central part" to that portion between the first and third planes; "anterior central part" to that portion between the third and fourth planes; "posterior frontal part" to that portion between the fourth and sixth planes; "median frontal part" to that portion between the fifth and sixth planes; "anterior frontal part" to that portion anterior to the fifth plane; "sphenoidal part" to that portion of the temporal lobe which is below a horizontal plane passing through the horizontal ramus of the Sylvian fissure.

This anatomical division, which will be founded on a more scientific basis as our knowledge of the subject increases, must be regarded as an extremely important step in advance; without it, all research would be comparatively useless. As it is, we possess very few data with regard to the localization of disease in this region of the brain, and our positive knowledge of the subject may be summed up in a very few words.

Small circumscribed foci, which are situated deep within the centrum ovale, will not give rise to psychical disturbances, when the foci occur singly. It has not yet been determined whether this remark also holds good with regard to foci which are situated near the convolutions.

True aphasia may be produced by affections of the centrum ovale. In these cases, the lesion was always situated in, or at least implicated, the "posterior frontal part," especially in its lowermost bundles of fibres, in the immediate neighborhood of the third frontal convolution. The lesion, furthermore, always involved the left hemisphere. Diseased processes, which are situated at some distance from the cortex, do not appear to be able to give rise to aphasia. There is some reason also to believe that disturbances of articulation may be caused by lesions of the "posterior frontal part." We are unable, however, at present to differentiate the speech-disturbances due to lesions of the centrum ovale from those occurring in cortical affections.

Nothing positive is known with regard to the occurrence of sensory disturbances in diseases of the centrum ovale; this is also true concerning disorders of the special senses.

The motor symptoms connected with diseases of the centrum ovale have been more thoroughly and satisfactorily investigated than the other morbid phenomena.

Affections of the "occipital part" are unattended with any motor symptoms. Lesions situated in the "median and anterior frontal parts" are also unattended with any symptoms of this character; this is also, in all probability, true of lesions of the temporal lobes, though this point has not yet been definitely determined.

When lesions of the centrum ovale give rise to motor paralysis, the affection is always situated between the limits of the "posterior frontal part" and "posterior central part." Perhaps further investigation will show that that the "posterior frontal part" must also be added to this motor region. The clinical history of paralysis due to lesions of this character may correspond in all respects to that due to affections of the corpus striatum, *i. e.*, a hemiplegia, affecting the face and limbs on the opposite side of the body. When this form of paralysis is produced, it will be impossible to differentiate it from that due to lesion of the corpus striatum, unless true aphasia is also present, when it is rendered certain that there must be an affection of the white fibres between the corpus striatum and the cortex cerebri. Secondary contracture may develop in the paralyzed limbs in the same manner as in diseases of the pons, cerebral peduncles, or internal white capsule. Early primary contracture is also occasionally observed and, according to *Charcot*, must be attributed to irritation of the caudate nucleus produced by the tearing-across of the white fibres of the centrum by the hemorrhage.

Joffroy has expressed the opinion that the occurrence of acute bedsores on the side opposite to the cerebral hemorrhage is connected with the localization of the lesion in the occipital lobe. This, however, has been disproven by more recent investigations.

CORPUS STRIATUM.

A hemorrhage of any considerable size which affects the corpus striatum will give rise to hemiplegia of motion upon the opposite side of the body. Not all the motor cerebral nerves are implicated, however; the following ones being unaffected, viz., the spinal accessory, pneumogastric, abducens, trochlearis, motor oculi communis, and the motor root of the trigeminus (nerve of mastication). *Nothnagel* claims that lesions of this character always cause paralysis of both limbs, in other words, that they do not give rise to monoplegia. The truth of this statement, however, has not been demonstrated.

Acute destruction of one lenticular nucleus produces motor paralysis, but chronic, stabile lesions in this locality may exist without paralysis. In other words, the paralysis which arises in consequence of a unilateral lesion of the lenticular nucleus is not permanent, but merely temporary. This was very well shown in a case recently under my observation, in which the patient had suffered at first from motor paralysis of the left arm and the left side of the face (with the exception of the orbicularis palpebrarūm and frontalis); there was also slight thickness of speech which persisted until death; the tongue was, however, freely movable in all directions. The paralysis of the face and arm gradually disappeared until the normal amount of power was restored. The autopsy revealed the remains of an old hemorrhage in the outer division of the right lenticular nucleus in its posterior third; it then skirted the lower border of the lenticular nucleus, and then passed upwards for a very short distance, implicating to a slight extent the posterior third of the internal capsule.

Hemorrhages into the corpus striatum produce the same effects as those in the lenticular nucleus, so that the clinical history will not enable us to differentiate between lesions of these two organs.

Destruction of the anterior portion of the internal capsule (between the corpus striatum and lenticular nucleus), gives rise to permanent hemiplegia on the opposite side of the body; similar effects are occasionally produced by lesions of the posterior portion of the internal capsule (between the optic thalamus and lenticular nucleus), in those cases in which the motor fibres contained in the latter are alone involved. A case has also come under my observation which supports *Nothnagel's* statement with regard to this mooted point.

When compared with the motor symptoms, sensory disturbances are rarely observed in lesions of the internal capsule. In exceptional instances, however, complete hemiplegia of sensation may be permanently produced, affecting all the forms of tactile sensation. In the majority of these cases, the special senses are also anæsthetic. The affection of sight is very peculiar, and may either consist of complete amaurosis or the field of vision may merely be narrowed upon all sides, thus differing in all respects from the hemiopia to which reference has previously been made. As in hysterical hemianæsthesia, the field of vision for the perception of colors is also narrowed and may even be entirely abolished. According to *Leber*, the amblyopia is bilateral, not unilateral, the eye on the side of the lesion being also affected, though to a less extent. The parts which must be involved in order to give rise to the symptoms just mentioned are the posterior part of the internal capsule and the adjacent part of the foot of the corona radiata. Whenever, therefore, there is profound anæsthesia of one-half of the body, coincident with special sense disorders, the lesion must be situated in the posterior part of the internal capsule or the adjacent part of the foot of the corona radiata.

As a rule, the hemianæsthesia is combined with hemiplegia, and only in exceptional instances does the latter disappear while the former remains permanent. Post-hemiplegic chorea and athetosis are regarded by *Charcot* and his school as due to lesions of the posterior part of the internal capsule and of the optic thalamus and caudate nucleus. In support of this opinion, *Charcot* states that hemianæsthesia is frequently observed in conjunction with hemichorea. Although a large number of cases of the latter affection have come under my observation, in not a single one have I noticed hemianæsthesia. *Gowers* attributes hemichorea to lesions of the optic thalamus. Finally, *Kahler* and *Pick* have advanced the opinion that the entire class of post-hemiplegic movements are due to irritation of the pyramidal motor fibres in any part of their course above the decussation of the pyramids. This question must, therefore, be regarded as still undecided.

THALAMUS OPTICUS.

We possess very little accurate and positive knowledge with regard to the effects of lesions of the optic thalamus. In the large majority of cases, it is doubtful whether the symptoms attributed to lesions in this locality are not due to remote effects upon distant parts. It is, therefore, very rarely possible to make a diagnosis of disease of the part in question.

Motor paralysis is never produced by lesions of the optic thalamus. It is highly probable, also, though this point is still sub judice, that they do not give rise to any sensory disturbances. It seems probable that, in those cases which are adduced in support of the view that disease of the thalamus produces anæsthesia, the adjacent portion of the internal capsule was also affected, and this part, as we have seen above, transmits sensory impressions from the periphery.

Lesions of the posterior third of the optic thalamus appear to give rise to visual disturbances in very rare instances. It is undecided at present whether these disorders are of the character of crossed unilateral amaurosis or of hemianopsia. It is evident, however, from the remarks which had previously been made with reference to cortical localization, that the visual disturbance in question will be of no assistance in diagnosis.

CORPORA QUADRIGEMINA.

From the observations hitherto made, it appears that the symptoms produced by lesions of the anterior and posterior pairs of the corpora quadrigemina are different. Lesions of the anterior pair seem to be always attended by partial or total blindness of both eyes. This symptom cannot, however, be considered characteristic of disease of the corpora quadrigemina unless an ophthalmoscopic examination furnishes negative results. Lesions of the posterior pair are accompanied by paralysis of the motor oculi communis; corresponding branches of both nerves are usually affected. Some observations appear to indicate that affections of the posterior pair are attended by disturbances of co-ordination, like those occurring in lesions of the cerebellum, to which we shall presently refer.

CEREBELLUM.

In not very rare cases, lesions of the cerebellum do not give rise to any morbid symptoms, and are accidentally found upon autopsy. In cases of this character, the morbid process is situated in one of the cerebellar hemispheres, and is always of such a nature that it produces no effects upon neighboring organs, such as the pons Varolii or medulla oblongata. *Nothnagel* is of the opinion that lesions, whose effects are restricted to one cerebellar hemisphere, cannot be diagnosed, but this view is contrary to that of various other authors, so that the question must be still regarded as sub judice.

Disturbances of co-ordination (tottering gait) and vertigo are characteristic of cerebellar disease, but these symptoms may also occur in other affections of the central nervous system, and cannot, therefore, be regarded as pathognomonic. The diagnosis must depend upon the combination of a number of positive and negative symptoms.

Nothnagel believes that the tottering gait and vertigo are always due to an affection of the middle lobe of the cerebellum; he acknowledges, however, that in exceptional instances the middle lobe may be implicated, although these symptoms are not observed.

Amblyopia and amaurosis together with mydriasis may occur in cerebellar hemorrhages, but it is still doubtful whether these symptoms do not depend upon secondary implication of the corpora quadrigemina. In the same manner, it is doubtful whether "forced movements" are due to lesions of the cerebellum proper or to a secondary affection of the cerebellar peduncles.

PACHYMENINGITIS HÆMORRHAGICA INTERNA.

BIBLIOGRAPHY.

FUERSTNER: Zur Genese und Symptomatologie der Pachymeningitis hæmorrhagica. Archiv f. Psychiatrie u. Nervenkrankhtn., 1878, Bd. VIII., p. 1-31.—BAUER: Drei Fälle von Pachymeningitis hæmorrhagica interna. Ann. d. städt. allg. Krankenh. zu München, 1878, I., p. 724-726.—MOSLER: Zur localen Behandlung der Hirnhaut-Affectionen. Deutsch. Archiv f. klin. Med., 1879, XXIV., p. 246-248.—LEDOUX: Hématome de la dure-mère. Progrès méd., 1880, VIII., p. 668.

Fürstner opposes the view of a primary extravasation of blood as the initial lesion in hæmorrhagic pachymeningitis. This opinion is based upon the fact that, in the most recent and delicate pseudo-membranes upon the dura mater, very slight traces of blood are found—a condition which is incompatible with the theory of an initial hemorrhagic extravasation. In addition, the latter view will not explain those cases in which small false membranes at the base of the skull are separated from one another by a very small tract of unaffected dura mater.

This author has also shed considerable light upon the symptomatology of the acute hemorrhages which occur at intervals in the disease under consideration.

When a pachymeningitic hemorrhage occurs in general paralysis of the insane or in delirium tremens, there is a sudden and enormous increase of the motor restlessness, and of the delirium; "the patients chatter continually in an incoherent manner, they throw the bed-clothes in all directions, make attempts at climbing, knock the head and limbs recklessly against the sides of the bed, make violent resistance to everything that is done; the face is strongly congested, profuse perspiration is noticeable, and the frequency of the pulse is increased." After this excitement (which is essentially different from that usually observed in delirium tremens, although it is very difficult to define its peculiarity) has lasted for some hours, the patient gradually becomes more quiet, appears to fall asleep, mutters to himself occasionally in a delirious manner; the respirations and pulse become slower, the face appears paler, and increasing somnolence develops. If the protracted somnolence, in cases of this character, is not associated with paralytic symptoms, we are justified in concluding that the symptoms are caused by an extra-cerebral hemorrhage. The latter may, however, be either traumatic, aneurismal, or pachymeningeal, and the differentiation of these conditions must depend upon the other circumstances of the case.

The development of the pachymeningeal hemorrhage does not, however, always occur in the manner above described. The scene is sometimes opened by the appearance of a series of epileptic seizures. These are either entirely unilateral at the onset, and remain so during the entire course of the disease, or after having been confined to one side for a certain length of time, they cease in this part of the body and affect the limbs on the opposite side, or, finally, they can, in no respect, be distinguished from ordinary bilateral epileptic seizures. The comatose condition develops during the intervals between the convulsions or after the latter have ceased.

In a much smaller number of cases, the clinical history begins with an attack of unconsciousness as in ordinary cerebral apoplexy. In such instances, however, the pachymeningitic hemorrhage is always very profuse, and these cases cannot, therefore, be differentiated from intra-cerebral hemorrhage.

The temperature of the body usually rises from the beginning of the attack, and may even attain 41° before the fatal termination results. When the disease runs a protracted course and presents remissions and exacerbations, the temperature varies in a corresponding manner. This initial rise of temperature is, to a certain extent, a differential feature which serves to distinguish it from intra-cerebral hemorrhage, in which, according to *Bourneville* and *Charcot*, there is an initial fall of the temperature. As *Huguenin* remarks, however, this latter statement does not always hold good, and I can substantiate this opinion from my own experience. In a case of cerebral hemorrhage to which I was called while the extravasation was taking place, the temperature began to rise from the very beginning, and continued so to do until death. In a certain proportion of cases of hæmatoma of the dura mater, the elevation of temperature is due to the coincidence of diseases of the pulmonary organs, but there is no doubt that this phenomenon also occurs in uncomplicated cases.

Certain features attending the comatose condition also present considerable importance from a diagnostic stand-point. As a rule, it begins gradually, and is often preceded by the motor irritative symptoms, to which attention has been previously directed; it also continues for a certain length of time, without being accompanied by any paralytic phenomena. The most characteristic feature of the coma, finally, is the occurrence of remissions and exacerbations. Patients who are in a state of complete unconsciousness at one time, will respond to various forms of irritation within a few hours, and may even answer and comprehend questions which are put to them. After a short period, however, they may again lapse into a state of profound coma. These variations in the condition of consciousness may be noticeable for several days.

Fürstner has also called attention to very interesting ophthalmoscopic appearances in the affection under consideration.

1. Choked disk may occur on both sides, although no extravasation has occurred into the sheath of the optic nerves. In this event the pachymeningitic hemorrhage has been sufficiently extensive to produce compression of the brain, and thus cause an escape of cerebro-spinal fluid from the subdural space into the optic sheaths.

2. Choked disk may occur on both sides, together with an extravasation of blood into the optic sheaths. These cases are of extremely rare occurrence, and only three have been hitherto reported.

3. Choked disk may occur on only one side. *Fuerstner* states that in all cases of pachymeningitic hemorrhages, in which unilateral or bilateral choked disk was observed during life, the autopsy always showed the presence of a unilateral or bilateral hemorrhage into the anterior and posterior cerebral fossæ, and clots of blood were usually found in the vicinity of the optic chiasm. The extravasation of blood, therefore, occurred at times into one, at times into the other optic sheath. The anatomical conditions, therefore, give rise to the comparatively frequent occurrence of unilateral choked disk (on the side opposite that of the paralysis), while in the general and uniform increase of pressure due to tumors, hydrocephalus, etc., bilateral changes are usually observed in the fundus of the eye.

4. *Huguenin* reports a case of extravasation into the sheath of one of the optic nerves without the development of choked disk. This observation is a unique one.

These appearances possess considerable importance, with reference to diagnosis from other cerebral lesions, which may also give rise to choked disk.

The conjugate deviation of the head and eyes, described by Prévost, is also noticed at times in variable degrees. In some cases it is observed for a very short while, especially at the beginning of the attack; in others it continues for a long time. The head can be readily moved from the "forced position" to the opposite side, in which it sometimes remains for a little while, but it gradually returns to the previous position.

Another interesting phenomenon described by *Fuerstner* is the peculiar nystagmus sometimes noticed, especially in combination with conjugate deviation of the head and eyes. "If, for instance, the right half of the body is affected by the paresis, the patient's eyes are often found to move to and fro for hours at a time, and always maintain the same limits from extreme outward deflection towards the right side back to the median line." If the somnolent patient is aroused and directed to look at an object held before him, the nystagmus ceases temporarily, and he is able to follow the object from the median line into the right field of vision, but not beyond the median line into the left field of vision; it is evident that the patient is endeavoring to look towards the left, but, after a few ineffectual efforts, the eyes become directed towards the extreme right, and the movements of nystagmus recommence. *Fuerstner* does not venture an opinion with regard to the cause of these peculiar movements, but he has observed them so constantly in pachymeningitic and other surface hemorrhages, that he regards them as a valuable auxiliary in diagnosis.

The motor disturbances also furnish valuable aids in diagnosis. Hemiplegia is rare, and only occurs in those cases of profuse pachymeningitic hemorrhage which can hardly be differentiated from intra-cerebral extravasations. As a rule, paresis gradually develops upon one side of the body. In some cases, it gradually becomes more marked; in others, it merely continues for a few hours. It is a peculiar fact that the grade of paresis may vary considerably within a short space of time, so that it presents distinct remissions and exacerbations. Slight contracture of various muscles is also noticeable, sometimes upon the paretic, sometimes on the unaffected side.

COMMOTIO CEREBRI.

BIBLIOGRAPHY.

DURET: Etudes expériment. et cliniques sur les traumat. céréb. Paris, 1878.

As the result of his admirable investigations into this subject, *Duret* has arrived at the following conclusions:

During a severe fall upon the head, or as the result of a blow upon the skull, a wave of cerebro-spinal fluid is formed around the cerebral hemispheres and in the ventricles, which distributes the violence received at one point to all parts of the central nervous system, and more especially in the region of the medulla oblongata.

The violent action of this wave of fluid usually produces its most serious and extended effects in the arachnoid lymphatic spaces at the base of the brain, around the medulla, and especially upon the floor of the fourth ventricle and upon the restiform bodies.

The skull, being very elastic (as is readily shown by the fact that it will rebound to a considerable height when allowed to fall upon the floor), becomes depressed at the point of injury, producing at this region a "cône de dépression." This is immediately followed by the formation of a "cône de soulèvement" at the opposite extremity of the axis of percussion. The wave of cerebro-spinal fluid naturally flows from the "cône de dépression" to the "cône de soulèvement," and then returns, thus producing a vacuum in the latter; the blood then rushes into the vessels of the latter region, in order to fill up this vacuum; they become distended and rupture, thus producing slight extravasations of blood, which accumulate in the meshes of the pia mater, or in the substance of the nervous tissue.

The production of the "cône de dépression" has been directly proven by experiment, but *Duret* has not advanced the slightest shadow of evidence in favor of his theory of the formation of a "cône de soulèvement." Such a phenomenon would be very difficult of demonstration by experimental means; but if we take into consideration the fact that the skull is filled by a substance of slight consistence, which is semi-fluid rather than solid, it appears much more probable that the force which is applied at one part of the skull will be distributed almost uniformly to the entire skull, and will not result in the formation of a "cône de soulèvement." Nevertheless, the "cône de dépression" is a potent factor in the production of the lesions observed in the condition under consideration. The wave of fluid produced in the ventricles by the formation of this "cône de dépression" is amply sufficient to explain the extravasations of blood, etc., which are so uniformly observed.

At the moment when the injury to the skull is received, an excess of tension is produced in the lymphatic sheaths which surround the blood-vessels of the brain; in consequence of this, pressure is produced upon the latter, and temporary anæmia of the entire brain is thus induced.

This anæmia is increased and prolonged by reflex vascular contraction, the point of departure of which resides in the irritation of the restiform bodies (to which we have referred above), and of all the sensory parts of the mesocephalon.

The general vascular contraction is followed by an equally extensive vascular paralysis, as the result of which there is a suspension of the nutritive changes between the blood and the constituents of the nervous tissues. As a consequence, the latter cannot resume their functions. Finally, the vascular paralysis sometimes continues until inflammatory reaction occurs, and the nervous symptoms may then continue until a fatal termination.

Duret believes that the clinical history of cerebro-spinal shock is made up by the combination of cerebral, bulbar, and spinal symptoms, the predominance of one or the other, and their duration, varying according to the severity of the injury, and also according to the portion of the skull at which the blow was received. In slight shock, the symptoms are temporary, and may only last a few moments. In severe shock, they vary in duration from a few minutes to several hours. The symptoms may be regarded as appearing in two periods.

The first period includes the spasmodic or tetanic, and the paralytic stages.

The spasmodic or tetanic stage is characterized by vascular spasm and general anæmia of the nervous centres.

The cerebral functions are suddenly abolished, *i. e.*, there is loss of intelligence, voluntary power of motion, and sensory perceptions. The functions of the medulla oblongata are also affected to a greater or less extent. This is evidenced by a more or less prolonged attack of cardiac and respiratory syncope; in addition, there is tetanic spasm of the muscles of organic and animal life, caused by irritation of the sensory fibres of the mesocephalon, and especially of the restiform bodies.

At this time, the central temperature rises; the pulse is bounding, the heart contracts forcibly, and the respiratory movements, which were temporarily suspended, are effected with difficulty, on account of the spasm of the muscles of respiration. This stage only lasts for a very short time, from a few seconds to several minutes.

The symptoms of the paralytic stage vary according to the severity of the injury sustained. Somnolence, sopor, or coma continue more or less marked, together with loss of the power of voluntary motion and of sensory perceptions. The bulbar symptoms consist of a secondary acceleration of the respiratory movements, which is followed, when grave lesions are present, by very marked retardation. The pulse may remain slow, but it is not full; the heart does not contract forcibly, the vessels throughout the body being in a state of relaxation, and thus offering but slight obstacle to the passage of blood. The central temperature now becomes reduced, as the blood, which in the previous stage had been forced into the abdominal viscera, now returns to the surface of the body.

The symptoms on the part of the spinal cord consist of complete loss of muscular power and of vascular tonus throughout the body.

The second period is known as that of congestive and inflammatory reaction, and is usually due to the presence of localized or diffused lesions (small extravasations of blood, rupture of the vessels or of the nervous tissues, rupture of capillaries into the lymphatic vascular sheaths). In this stage the coma continues or is succeeded by somnolence and sopor; the sensibility of the entire body remains obtuse; movements are performed unconsciously, and only when the patient is roused by strong stimuli. The bulbar symptoms consist of a febrile condition of the pulse and respiration, and of an elevation of temperature.

In *foudroyant* shock, death occurs from sudden anæmia of the medulla oblongata, either in consequence of the excessive pressure which is produced in the cerebro-spinal fluid, or on account of the violence of the reflex contractions of the cerebral vessels. In these cases, the respiratory and cardiac syncope, which is temporary in the two other varieties of shock, proves fatal.

The site at which the violence is received possesses great influence upon the character of the morbid phenomena produced thereby.

When the blow is received upon the forehead, its effects are chiefly manifested at the base of the brain, the pons Varolii, and medulla oblongata, and sometimes in the spinal cord. In blows upon the side of the head, lesions are produced in the opposite cerebral hemisphere. In this event, the symptomatology is somewhat peculiar, and may be designated as "hemisphere shock." Although injury is always sustained by the medulla oblongata in these cases, as is evidenced by the temporary arrest of the cerebral circulation, and by the cerebro-medullary disorders, unilateral disturbances, such as unilateral contracture, propulsion, or forced movements are not rarely observed at the moment of injury. If grave lesions have been produced, hemiplegia or hemianæsthesia may occur at a later period; these disorders occur upon the same side as the seat of the injury, since the cerebral lesions are produced upon the opposite side. In blows upon the occiput, the lesions occur in the frontal lobes, in the posterior parts of the hemispheres, and in the medulla oblongata. In blows upon the neck, the medulla oblongata may be affected by the return wave of cerebro-spinal fluid. Each variety of shock, therefore, usually produces special lesions, according to the site of the injury received.

In very exceptional instances, meningitis may follow injuries to the head, although no lesion can be detected as the direct result of the violence. This is proven by the history of the following case reported by *Forget:*

A boy, æt. 17 years, fell a distance of six feet, striking probably on his head. He did not lose consciousness, returned home, and ate and slept well. Fifteen hours after the receipt of the injury, he began to suffer from headache, which was soon followed by unconsciousness and automatic movements; the pulse was seventy-two, the respirations labored. No external violence could be discovered. During the following night, tetanic convulsions developed and death soon ensued, the entire course of the disease lasting thirty-six hours. At the autopsy, thirty grammes of pus were found upon the convexity of the brain on the right side; no fracture or contusion could be discovered, and the brain was intact.

ACUTE ENCEPHALITIS IN PYÆMIA.

Huguenin has observed this form of encephalitis in the following diseases :

1. *In ulcerative endocarditis:* In this form, the abscess is due to emboli which have been carried from the heart to the vessels of the brain. *Heschl* has observed embolism of various cerebral arteries in this condition, although no plugs could be found in those vessels which were in the immediate neighborhood of the abscesses.

2. *In mycotic endocarditis:* This form is of rare occurrence, but there is no doubt with regard to the mode of formation of the abscesses. *Eberth* has found micrococci in the vessels of the brain, similar to those on the valves of the heart. We must, therefore, regard the cerebral infarctions and secondary abscesses as due to multiple emboli of a mycotic character.

3. *In mycotic osteomyelitis:* Several cases were observed complicated with pulmonary abscesses, in which micrococci were found in various localities. There were also multiple infarctions and abscesses in the brain. *Eberth* found micrococci in numerous cerebral vessels, so that this form may be regarded as similar in character to those previously mentioned.

4. *In the pulmonary gangrene of bronchiectasis:* This is illustrated by the following case:

A man, æt. 21 years, was admitted to hospital on Oct. 17th, 1877, and died on Oct. 19th. No previous history could be obtained. The patient is delirious and incoherent, and does not know where he is. Considerable cough and expectoration of clear, red blood; temperature 39°, pulse 104. Pupils of normal size and react well; the patient states that he can hardly see, cannot count the fingers; complains of severe headache. Tongue very dry, speech and audition normal, no rigidity of the neck, no paralysis, convulsions, "boat-belly" or meteorism; the urine does not contain albumen. Vesicular breathing in apices of lungs, coarse rhonchi extending from infraclavicular space to lower borders of lungs. Posteriorly there is slight dulness on the right side from the spine of the scapula to lower border of the lung; numerous semi-metallic, moist râles of moderate size; no crepitation or bronchial breathing. Left retina: fundus pale, veins very tortuous; the optic nerve shows a partial neuritic swelling to the inside and upwards.

Oct. 18th. Condition unchanged. At 10 A.M., a severe chill. Patient entirely unconscious, tremor of the right arm at times; no paralysis or convulsions. Three P.M.; another severe chill; expectoration of very fœtid reddish-brown sputum. Evening temperature 38.6°, pulse 112.

Oct. 19th. A convulsive seizure during the night; another seizure at 3 o'clock. Death.

Autopsy.—Dura mater normal; five to six hemorrhagic infarctions, some containing pus in the centre, are situated in the cortex of the right temporal lobe. Small extravasations into the pia mater over the right frontal and parietal lobes, and a few small vessels in the pia mater are filled by yellowish-brown masses (fat and detritus). Purulent hemorrhagic infarctions in the right convexity, and also in the right corpus striatum.

Bronchiectasis with broncho-pneumonia in the vicinity, which are undergoing a gangrenous process. According to *Huguenin*, the embolic masses found in the vessels of the pia mater were derived from the gangrenous foci in the lungs.

GENERAL PARALYSIS OF THE INSANE.

(*Dementia Paralytica.*)

BIBLIOGRAPHY.

Meyer, Ludwig: Die Behandlung der allgemeinen progressiven Paralyse (Dementia paralytica). Berl. klin. Wochenschr., 1877, 21, p. 289.—Tuke: The Diagnosis of general Paralysis of the Insane. Edin. Med. Journ., 1877.—Krafft-Ebing: Zur Kenntniss des paralytischen Irreseins beim weiblichen Geschlechte. Arch. f. Psych. u. Nervenkrankhtn., VII., S. 182.—Leidesdorf: Klinischer Beitrag zur paralytischen Geistesstörung. Oesterr. med. Jahrbücher, Heft II.—Voisin: Traité de la paralysie générale des aliénes, Paris, 1879.—Foville: Contribution à l'étude des rapports entre la syphilis et la paralysie générale. Annal. méd. psych., 37 année.—Mickle: General Paralysis of the Insane. London, 1880.—Haunhorst: Zur Behandlung der progessiven Paralyse. Berl. klin. Wochenschr., Mar. 29th, 1880, p. 182.—Meyer, Ludwig: Ueber die Behandlung der progressiven Paralyse durch Scheitelfontanellen. Berl. klin. Wochenschr., April 12th, 1880, p. 211.

Ludwig Meyer strongly recommends the adoption of the following method of treatment, in dementia paralytica: In the region of the large fontanelle the scalp is shaved with a razor over a surface half as large as the hand, and tartar emetic ointment rubbed into the centre, over a space as large as a dollar. The first inunction should be very carefully and thoroughly performed. A portion of ointment about as large as a pea is rubbed in with the aid of a little wad of linen, and the surface is then covered with linen which has been smeared with ointment. The second inunction is performed after the lapse of twenty-four hours, and friction should be made very carefully in order to avoid tearing off the loosened epidermis. The denudation of the cuticular layer causes distressing pains and is not advantageous, since our endeavor should be to remove the parts as deeply as possible. Two inunctions are usually found to be sufficient. For the sake of precaution, a small quantity of ointment is applied, without inunction, to the surface on the third day and, if necessary, a cloth smeared with it may be kept constantly applied. During the course of the third and fourth days, the swelling increases and spreads to the frontal region and even to the face. In this method of application, the pustular eruption attains very slight proportions. As soon as the surface presents a marked degree of swelling, it should be kept covered with warm poultices. In a few days almost the entire portion of skin which has been subjected to inunction is removed by suppuration; those parts which are still adherent, may be touched with caustic potash or removed with a pair of scissors. The entire procedure occupies about two weeks until its completion and leaves a deep, suppurating sore which may be kept open for two or three months by unguent.

basilic. During this entire period, iodide of potassium is administered internally in moderate doses (potass. iodid., 3.–5.0; aq. destil., 180.0. one tablespoonful four times a day).

Of fifteen cases treated in this manner, eight were cured. One patient who desisted prematurely from treatment, had a relapse after an interval of two years; in another, the mental condition remained intact after the lapse of three years; in four others, *Meyer* has remained in communication with the patients who are still well.

It should be remembered that all these patients began treatment in the early stages of the disease.

Voisin has obtained the best results in this disease from the use of cold baths. In cases which are attended with considerable rise of temperature, the water should have a temperature of 12° C., and the patient be kept in it for ten minutes. When the fever is trifling in degree, the baths should, in the beginning, be at 18° C., and last for five minutes; at the end of four to six days, the patient may be kept in the bath for ten minutes, and, after the lapse of a fortnight, the temperature may be reduced to 12°. In winter, the baths should be given in rooms thoroughly heated. After being taken out of the water, the patient is wrapped in warm blankets, in which he is kept from three quarters of an hour to an hour, and then thoroughly dried. This procedure should at first be repeated daily and, after a while, on alternate days; it may be kept up with benefit for a very long period. The baths should not be given during the menstrual period, nor unless the patient is under our direct supervision at the time of the administration.

DISEASES OF THE MEDULLA OBLONGATA.

BIBLIOGRAPHY.

ROBINSON, CH. H.: Bulbar Paralysis. Lancet, Oct. 12th, 1878.—LEYDEN: Ueber progressive amyotrophische Bulbärparalyse und ihre Beziehungen zur symmetrischen Seitenstrangsclerose. Archiv f. Psychiatrie, Bd. VIII.—GRASSET: Deux observations de paralysie labio-glosso-laryngée, l'une primitive, l'autre secondaire.—Abolition du goût dans un cas. Montpell. méd., Juin.—WILLIGK. Ueber apoplectiforme Bulbärparalyse. Deutsch. Arch. f. klinische Medicin, Bd. XXII., S. 101.—QUÉNU: Tubercle du cerveau de la protuberance et du bulbe. Progrès méd., 1878, VI., 881.—EULENBURG: Lehrbuch d. Nervenkrankhtn. Berl., 1878. — FISCHL, J.: Zur Casuistik d. acuten Bulbärparalyse. Prag. med. Wchnschr., 1879, IV., 33–53.—SMITH, J. G.: Hemorrhage into Pons and Medulla. Trans. Bristol Med.-Chir. Society, 1878, 115–117.—DE WITT, W. H.: Glosso-laryngeal Paralysis. Cincin. Lancet and Clinic, 1879, n. s., II., 184.—ERB, W.: Zur Casuistik d. bulbären Lähmungen. Arch. f. Psychiat., 1879, IX., 325–350.—

Grasset, J.: Maladies du système nerveux. Montpel., 1879.—Minot, F.: Bulbar Paralysis. Boston Med. and Surg. Journ., 1879, c. 432–434. Weiss: Ueber progressive Bulbärparalyse. Wien. med. Wchnschr., 1879, XXIX., 929-931. — Bauduy: Labio-glosso-pharyngeal Paralysis. St. Louis Cour. of Med., 1879, II., 443–447.—Lelion: Ramollissement bulbaire, paralysie des membres et des nerfs bulbaires. Gaz. hebd. de méd., 1879, 2 s., XII., 558.—Kahler and Pick: Ein Fall amyotrophischer Bulbärparalyse mit den Erscheinungen d. amyotrophischen Lateralsklerose. Prager Vrtljschr. f. d. prak. Heilk., 1879, II., 72-83.—Dowse: Remarks on Bulbar Paralysis with Special Reference to Artificial Feeding. Brain, 1879, II., 191–202.—Eisenlohr: Ueber acute Bulbär- u. Ponsaffectionen. Arch. f. Psych., 1879, X., 31–47.—Lubanski: Note sur un cas de tumeur du quatrième ventricule. Union méd., 1879, 3 s., XXVIII., 1034–1038.—Bergerhof: Ein Fall v. Tumor d. Corpus quadrigem. m. acuter Bulbärparalyse, 1879.—Schaaf: Ueber progressive Bulbärparalyse, 1879. — Duval and Raymond: Paralysie labio-glosso-laryngée. Arch. de Physiol. norm. et path., 1879, 2 s., VI., 735–750. — Müller: Zur progressiven Bulbärkernlähmung. Oesterr. ärztl. Vereinsztg., 1880, IV., 3-5.—Pitres and Sabourin: Note sur un cas de paralysie labio-glosso-laryngée protopathique. Arch. de Physiol. norm. et path., 1879, 2 s., VI., 723–734.—Adamkiewicz: Ein Fall v. amyotrophischer Bulbärparalysie m. Degeneration d. Pyramidenbahnen. Charité-Annalen, 1880, V., 353–375.—Eisenlohr: Klin. u. anat. Beiträge z. progressiven Bulbärparalyse. Ztschr. f. klin. Med., 1879-80, I., 435–460. — Kirchhoff: Cerebrale glosso-pharyngo-labial Paralyse mit einseitigem Herd. Arch. f. Psych., Bd. XI., Nr. 1, p. 132–137.

Leyden, basing his opinion upon the autopsical examination of five patients suffering from "progressive amyotrophic bulbar paralysis," believes that this disease is always accompanied by a symmetrical, systematic sclerosis of the lateral columns of the spinal cord, which is combined with atrophic changes in the large multipolar cells in the anterior horns of the gray matter of the cord. "The anatomical substratum of the affection is a chronic, finally very diffuse degeneration of the motor paths, from the terminal apparatus in the muscles themselves through the motor nerves to the motor paths of conduction and the trophic centres in the spinal cord." This view is combated by *Pitres* and *Sabourin*, who, in a typical case of labio-glosso-laryngeal paralysis, found, upon autopsy, atrophy of the bulbar nuclei, although the spinal cord was found to be intact. In a case of a similar nature, *Duval* and *Raymond* found the white columns of the cord unaffected, but the large motor cells of the anterior horns were atrophied in part.

A few cases have been reported in which the symptomatology of labio-glosso-laryngeal paralysis was due to bilateral lesions of various portions of the cerebrum. *Kirchhoff* has recently reported an observation in which the clinical history of bulbar paralysis was produced by a unilateral lesion. The patient in question was suddenly seized with vertigo, severe pain in the right temporal region, and spasmodic twitchings in the arms and legs. The power of speech was at once lost, and he became unable to swallow; consciousness was retained. The saliva flowed constantly from the mouth. On the following day, the power of speech and deglutition had entirely returned, and the patient went back to work.

A week later, he had an attack of vertigo, fell suddenly to the ground, but was able to drag himself to bed. He was unable to swallow during the entire night, but the power of deglutition returned next morning; speech was somewhat hesitating; the saliva dribbled from the mouth; locomotion unaffected. The following condition was found upon admission to the hospital, the patient complaining chiefly with regard to the difficulty of speech: The two halves of the face are usually normal, but occasionally the right, and shortly afterwards the left, angle of the mouth are drawn upwards; the patient often laughs without cause. The naso-labial folds are poorly developed on both sides. He is unable to whistle; closure of the mouth and biting are unaffected; lateral movements of the mouth very slight. The tongue can barely be protruded one centimetre beyond the mouth. Difficulty of speech, especially in labials and laryngeal sounds; delay in closure of the glottis. The symptoms underwent variable changes for the worse, until five months after admission to the hospital, when he suddenly fell to the ground. The face was found to be drawn to the right; paralysis of left arm and leg; death ten days later. The autopsy revealed embolism of the right middle cerebral artery; the posterior two-thirds of the right corpus striatum presented a yellowish-white color; the underlying portion of the internal capsule of a transparent gray. The outer third of the nucleus lentiformis was softened, but not discolored; this softening spot grows larger posteriorly. The spot in the lenticular nucleus was inclosed by a firm, sclerotic wall, upon which a vascular membrane was found. Microscopical examination of the pons and medulla gave entirely negative results.

The glosso-labio-pharyngeal paralysis, with its bilateral symptoms, must therefore be attributed to the lesion of the lenticular nucleus. *Kirchhoff* states that this remarkable condition may perhaps be explained under the following hypothesis: "Both sides of the body are represented in each hemisphere; usually, however, only the system of fibres of the opposite half of the body is employed. It is, therefore, only necessary to assume that through some chance, either of development or more probably by the unilateral growth of the function, only the system of fibres passing from one hemisphere had assumed physiological conduction; their interruption would, therefore, lead to an abolition of the functions which otherwise depend on both hemispheres." This theory, it is evident, is extremely vague, and is scarcely worth discussion.

CONCERNING A NEW, PROBABLY BULBAR COMPLEX OF SYMPTOMS.

Under this heading, *Erb* calls attention to a new affection which may be regarded as a peculiar variety of bulbar paralysis. Three cases have come under this observer's notice, which presented a remarkable uniformity in the symptoms. The following is a short abstract of the history of one of these patients:

G. F., æt. 59 years; always enjoyed good health previous to present illness. Was taken sick in February, 1868, with shooting pains in the neck, increasing on motion; he frequently had slight attacks of headache.

In the middle of April, the patient noticed that he could no longer keep the head erect readily; this weakness of the neck increased, so that finally he could only raise the head with great difficulty.

In the beginning of June, he noticed a decided diminution in the power of the muscles of mastication; at the same time the upper eyelids became paretic, so that the eyes were opened with difficulty. For the past two or three weeks it was noticed that the tongue is moved with greater difficulty, and that the linguals are pronounced more poorly than the other consonants. Some disturbance has also been recently experienced in swallowing. In other respects the patient is entirely well.

Present Condition.—The patient, who is fairly well nourished, holds his head in a peculiar position: it either rests upon the chest, or is thrown so far backwards that it passes behind the line of gravity, and is only kept in position by the anterior muscles of the neck. The neck is somewhat flattened on both sides, and the bellies of the various muscles cannot be distinctly felt in this position, as they are evidently atrophied. The eyes are half closed. The superficial facial muscles present no abnormalities; the masseters soon relax their hold when the patient is directed to clinch the teeth; the mouth is usually kept open. The velum palati is moved somewhat more vigorously on the left side than on the right. The tongue can be protuded in a normal manner, but shows some signs of fibrillary twitchings.

There is slight diminution of the faradic excitability of the trapezii, masseters and splenii, but no other noticeable changes in their electrical excitability. There is distinct galvanic hyperæsthesia of the left acoustic nerve. A slight degree of paresis of the upper limbs was noticed.

The patient was treated by the application of the galvanic current in *loco morbi.* He slowly improved under this plan, and was discharged from the hospital on December 7th, 1868, almost cured.

The two remaining cases presented, in almost every particular, a striking resemblance to the one just described. An analysis of the symptoms shows that the parts chiefly involved are the motor oculi communis, the motor portion of the trigeminus, the spinal accessory, and the upper cervical nerves; and those more slightly affected are the facial nerve (the upper branches to the face), the hypoglossal, and probably also the glosso-pharyngeal nerves. The nuclei of origin of these nerves are all situated in the floor of the fourth ventricle and in its immediate neighborhood in the pons Varolii, and we have therefore to deal, in all probability, with a bulbar affection, which is entirely distinct from progressive bulbar paralysis. *Erb* supposes that, in the affection under consideration, the lesion is situated in the upper half of the fourth ventricle, involving the nuclei of the motor oculi communis, the motor root of the trigeminus, and the facial nerves, and he conjectures that the implication of the remaining nerves may be due to the spread of the lesion more deeply into the substance of the medulla, affecting their fibres as they pass upward from the nuclei of origin situated in the floor of the fourth ventricle.

DISEASES OF THE SPINAL CORD.

BY

R. VAN SANTVOORD, M.D.,

OF NEW YORK,

VISITING PHYSICIAN TO THE WORK-HOUSE AND ALMS-HOUSE HOSPITALS.

DISEASES OF THE SPINAL CORD.

ANATOMY OF THE SPINAL CORD.

BIBLIOGRAPHY.

AXEL KEY U. RETZIUS:—Studien in d. Anatomie des Nervensystems. Virchow-Hirsch, Jahresb., 1870, I., S. 28. Arch. f. mikrosk. Anat., IX., S. 308, 1873. Stud. in d. Anat. des Nervensyst. und des Bindegewebes. Grossfolio. Stockholm, 1875 und 1876, I. u. II.—EICHHORST: Entwicklung des mensch. Rückenmarks. Virch. Arch., Bd. 64, S. 425. — KRAUSE, W.: Allgemeine und mikroskop. Anatomie. Hannover, 1876.—SCHIEFFERDECKER: Regeneration, Degeneration u. Architectur des R.-M. Virch. Arch., Bd. 67, S. 542.—FLECHSIG, P.: Die Leitungsbahnen im Gehirn u. R.-M. des Menchen. Leipzig, 1876.—Ueber Systemerkrankungen des R.-M. Arch. d. Heilkunde, Bd. XXIII. u. XIX., 1877.—LÖWE, S.: Ueber d. Bindesubstanz im Centralnervensystem der Säugethiere. Arch. f. Psych. u. Nerv., XII., S. 1, 1876.—MAYSER: Experim. Beitrag z. Kenntniss des Baues u. s. w. *Ibid.*, VII., S. 599, 1877.—HUGENIN, G.: Anatomie des centres nerveux. Paris, 1879.—UNGER, L.: Ueber den Bau der grauen Substanz des centralen Nervensystems. Allg. Wien. med. Ztg., 1879, S. 491.

Axel Key and *Retzius* describe the arachnoid as a membrane which lies pretty close to and in parts, especially in the cervical portion of the cord, is adherent to the dura mater. Anteriorly, as far round as the ligament. dentic., the space between it and the cord, the subarachnoidal space, is crossed only by a few fibres. Posteriorly, this space is divided up by a longitudinal septum, incomplete in many places, and by numerous parallel and oblique fibres and membranous septa. These fibres and septa are made up of fibrous connective tissues, and are covered with endothelium. The arachnoid itself is composed of layers of fibrillary network, and is covered with a layer of endothelium. The pia is covered with a subarachnoidal fibre-network, in which the larger vessels are located.

Attention is called to the asymmetry which so often exists between the different sides of healthy cords. According to *Boll, Key* and *Retzius,* the pia sends in funnel-shaped prolongations with the vessels, which are continued over the capillaries, and form a system of intercommunicating *lymph-spaces,* which open into the subarachnoidal space. The subarachnoidal, as well as the subdural space, is in free communication with the sheaths of the nerve-roots and the lymph-vessels of the peripheral nerves. The subarachnoidal space of the cord communicates freely with that of the brain.

Unger concludes that the fine, so-called nerve-fibre network discovered by *Gerlach* in the gray matter of the cord is really connective tissue; because, in the chick, it appears before any ganglion cells are visible.

Carrière has proved the existence of the *anastomoses between the multipolar ganglion cells.* (*Arch. f. mikrosk. Anat.*, XV., S. 125, 1877.)

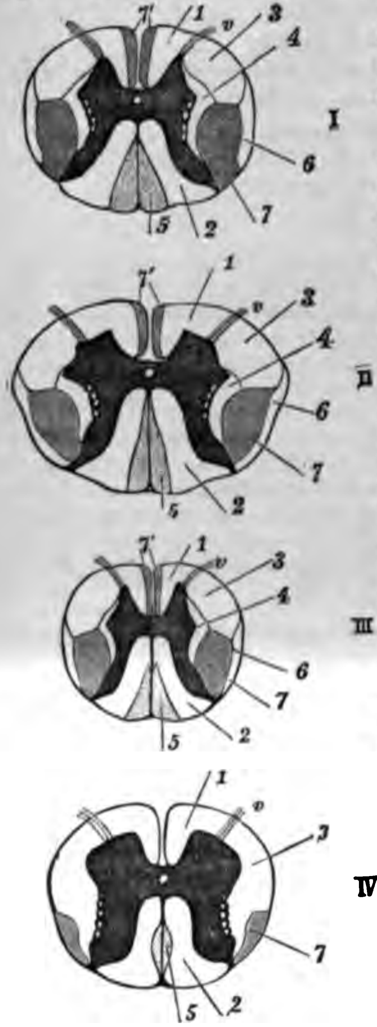


Diagram of the Developmental Systems of the Spinal Cord after Flechsig.

I, section at height of the 3d, II at height of 5th cervical nerves; III, at height of the 6th dorsal, and IV of the 4th lumbar pairs. 1, principal mass of the anterior columns; 2, wedge-tracts; 3, anterior mixed region of the lateral columns; 4, lateral boundary layer of the gray substance; 5, columns of Goll; 6, direct cerebellar lateral column-tracts; 7, pyramid tracts of the lateral columns; 7', pyramid tracts of the anterior columns; *v*, ante-[illegible] roots.

Mayser has demonstrated that some fibres of the anterior roots pass through the anterior horns to the gray substance of the opposite side.

The most important recent contribution to the anatomy of the cord is that of *Flechsig*. By the study of the development of the central nervous system in numerous fetuses and in children, he discovered that certain tracts in the white substance of the cord develop at different periods. From this circumstance he was able to divide the white substance into certain tracts or *systems*, which in the fully developed cord are, for the most part, no longer anatomically separable. These, in the order of their development, are first, the anterior columns exclusive of their inner fourth (*principal mass of the anterior columns*), and the posterior columns exclusive of the columns of Goll (*wedge-tracts*); second, the anterior halves of the lateral columns (*anterior mixed region of the lateral columns*); third, the layer of fibres lying next to the lateral periphery of the gray matter (*the lateral boundary layer of the gray substance*); fourth, *the columns of Goll*); fifth, a thin layer of fibres at the periphery of the lateral columns, reaching from or near the posterior nerve-roots to about the middle of the lateral columns (*the direct cerebellar lateral column tracts*), and sixth, the inner fourth of the anterior columns (*pyramid tracts of the anterior columns*), and a mass of fibres, including the posterior half of the lateral columns, except the third and fifth (*the pyramid tracts of the lateral columns*).

The pyramid and cerebellar tracts, and probably the columns of Goll, unite the nerve-centres above the cord with the different centres, etc., scattered at different heights along the cord. The principal mass of the anterior columns, the wedge-tracts, and the anterior mixed tracts of the lateral columns, for the most part unite the gray matter with peripheral organs, or unite different parts of the gray matter which lie at different heights on the cord.

The anterior pyramid tracts appear usually at the height of the lower dorsal vertebræ (variable), increase gradually in size, and finally pass upward into the pons without crossing over.

The lateral pyramid tracts appear in the lower half of the lumbar enlargement, and increase in size on going upwards. In the lumbar portion they are peripheral, but soon the cerebellar tracts appear, and separate them from the periphery, and they approach nearer to the gray substance. In the cervical portion they touch the periphery for a short distance again. In the pons each crosses over to the opposite side in the anterior pyramid.

The pyramid tracts are probably made up of fibres from the gray columns, which go either directly into the lateral columns of the same side, or through the anterior commissure, into the anterior pyramid columns of the other side, and are to be regarded as *indirect* continuations of the anterior roots—indirect, because interrupted by the ganglion cells.

The relative size of the pyramid columns varies. Usually the greater number of pyramid fibres are to be found in the lateral columns. Sometimes almost *all* the pyramid fibres are found in the lateral columns; sometimes almost all pass into the anterior columns. In the same individual most of the fibres of one side of the pyramid may cross over to the lateral column of the opposite side, while most of the fibres of the other side may not cross at all, *i. e.*, may pass into the anterior column. It is obvious how this irregular crossing may give rise to the asymmetry of the cord mentioned above. The exceptional non-decussation of the fibres is important in explaining the exceptional symptomatology of some lesions of the cord and brain (hemiplegia on the same side as the brain lesion).

The *direct cerebellar lateral tracts* appear in the upper part of the lumbar enlargement, partly as a compact bundle of fibres at the periphery of the posterior half of the lateral columns, partly as a number of isolated fibres scattered over its section. They increase in size in passing upward. They receive large bundles of fibres from the region of Clark's columns, and seem to be connected with these tracts and with the cells of Clark's columns (*Pick, Centralblt.*, 1878, No. 2). These tracts pass through the restiform bodies into the cerebellum.

The portion of the lateral columns not included in the above varies in size in different parts of the cord, corresponding to the size of the nerve-roots which enter at any given point. The connections and functions of the *lateral boundary layer of the gray matter* are little understood. The fibres of the *anterior mixed region of the lateral columns* come in part from the lateral parts of the anterior gray columns, in part they are to be regarded as the direct prolongations of the anterior roots. Their fibres pass in part back into the gray matter, in part into the medulla. Their function is wholly unknown.

The principal masses of the anterior columns do not increase in size from below upward. They come in part from the anterior roots, in part from the gray matter of the cord, and pass only in part directly into the medulla. Nothing more is known of them.

The columns of Goll are very small in the lumbar region; they increase steadily from below upward in size. Their fibres come partly from the posterior gray columns, partly from the posterior commissure. They terminate apparently in nuclei in the medulla.

The *wedge-tracts* vary greatly in size in different parts of the cord, increasing markedly at the enlargements. They are made up for the most part of direct continuations of the posterior roots, but there are also numerous longitudinal bundles of fibres in them. They terminate for the most part in nuclei in the medulla, but they enter also into the formatio reticularis, the olivary bodies, etc.

PHYSIOLOGY OF THE SPINAL CORD.

BIBLIOGRAPHY.

BALIGHIAN: Beiträge zur Lehre von der Kreuzung der motorischen Innervationswege im Cerebro-spinalsystem. Eckhardt's Beitrag zur Anat. u. Physiol., VIII., Bd., S. 193.—LUCHSINGER: Zur Kenntniss der Functionen des Rückenmarks. Arch. f. Physiol., Bd. XVI., S. 510.—SCHIFF: Ueber die Leitung der Gefühls-Eindrücke im Rückenmark. Allg. Wien. med. Ztg., 1879, S. 455.—KOCH, W.: Ein Beitrag zur Lehre der Hyperæsthesie. Virchow's Arch., Bd. 73, S. 273, etc.

The more recent investigations of *Schiff* confirm the observations of other writers, that when only a small portion of the gray matter of the cord remains intact, all the white matter and part of the gray having been severed, sensation to touch and pain remain in the parts posterior to the section. If, however, the intact portion is small, and in the extreme lateral periphery of the gray substance, sensation is retained on only one side of the body, together with a slight power of voluntary motion. The existence of different tracts for the different kinds of sensation has not been demonstrated, although *Brown-Séquard,* from certain facts in pathology, believes that they do exist. *Vulpian* denies their existence, believing that the different kinds of sensation only modify the sensitive tracts in different ways. *Schiff* confirms the observations of *Woroschiloff*, in that he proves that the white longitudinal fibres of the anterior lateral columns, although having chiefly to do with voluntary motion, "conduct a trace of sensation to the brain." *Woroschiloff* states that each lateral column contains sensitive fibres from both legs, the greater number crossing over to the opposite side of the cord. As the experiments were conducted only on the lumbar portions of the cords of rabbits, *Erb* is not inclined to accept the results unconditionally. The complete decussation of sensitive fibres is not yet proved. *Koch* confirms the experiments of *Ludwig* and *Woroschiloff*, who found that section of the inner two-thirds of the middle portion of the lateral columns in the lumbar portion of the cord gave rise to hyperæsthesia of the parts lying below, and on the same side as the section. The fasciæ, periosteum, and joint-surfaces shared in this hyperæsthesia. In the cervical portion of the cord and in the medulla, section of the *outer* fibres of the lateral columns gave rise to this phenomenon. Section of the outer portion of this region gave rise to hyperæsthesia of the joints alone, cutting the inner, of the skin alone; but in neither case was the hyperæsthesia so marked as when the whole region was severed. Strong electrical currents caused a cessation of the hyperæsthesia.

From the recent investigations of *Flechsig*, it seems probable that the pyramid-tracts are the chief conductors of the voluntary motor impulses. *Luchsinger* has proved that under the influence of picrotoxin convulsions would occur in the extremities behind the section of the cord, proving that, for this poison at least, the *convulsion centre* is not exclusively in the medulla.

The following summary of the present theories concerning the *innervation of the blood-vessels* is taken from *Erb's* second edition, no modification of it being necessary from more recent publications. References and authorities must be omitted for want of space.

There are probably ganglion cells on the vessels, or in their neighborhood, which serve as local vaso-motor centres. These local centres are under the influence of the larger nerve-centres, and are connected with them through two varieties of fibres, which lie in the peripheral nerves, viz., the vaso-constrictor and the vaso-dilator fibres. These fibres connect with two varieties of vaso-motor centres, which are scattered throughout the cord, and perhaps the upper nerve-centres, possibly also throughout the cerebral convolutions; but the most important are in the medulla oblongata. The two varieties of centres are the vaso-constrictor and the vaso-dilator centres. Both kinds of centres may be directly irritated, the condition of the vessels depending on which is most so. The constrictor nerves seem to need a stronger irritation than the dilator, in order to react. After section of the nerves, their irritability sinks more rapidly than that of the vaso-dilator fibres. Reflex influences may cause dilatation or contraction of vessels. Vessel-reflexes from the spinal centres certainly occur, but they are more limited than those radiating from the medulla. Similar reflexes may be excited from the local centres, manifested by dilatation of the vessels when the irritation is slight, by contraction when it is powerful.

The location of these centres in the cord is unknown. The vaso-motor nerves, as they come from the spinal centres, lie, for the most part, in the lateral columns. How they make their exit from the cord is, for the most part, unknown. The vaso-motor nerves for the head come from the cervical portion of the cord, those for the upper extremities from the thoracic portion, those for the pelvis and legs from the lower dorsal and lumbar portions. The vaso-motor nerves of the sciatic nerve, however, do not pass into it through the sacral roots, but pass through the sympathetic. A similar condition of affairs seems to exist in the brachial plexus.

Tschirjew confirms by elaborate experiments the reflex nature of the *knee phenomenon* (*Berl. klin. Wochenschr.*, 1878, No. 17). *Gowers* proved that *ankle-clonus* resulted from direct stimulation of the muscle, the time between the tap on the muscle and the reaction being only .4 to .3 of a second, too short a time for a reflex.

Nothnagel (*Arch. f. Psych.*, VI., S. 832, 1876) and *Lewinski* (*Ibid.*, VII., S. 327, 1877) have proved that strong irritation of distal parts (skin nerves) may exert an *inhibitory action* on tendon reflexes. *Erb* has proved the same fact in a case of spastic paralysis. In *Stümpell's* experience, flexion of the great toe has no inhibitory influence on ankle-clonus, as stated by *Brown-Séquard*. Flexion of the whole foot, by relaxing the tendo Achillis, causes its cessation. *Langendorff* has proved that the brain has a crossed action in inhibiting reflex action, the right side of the body being under the influence of the left side of the brain, etc.

Goltz, *Luchsinger*, *Ostroumoff* and *Nawrocki* have proved the existence of nerves which pass through the peripheral nerve-stems, and which, on irritation, cause a secretion of sweat. The centres for these nerves lie chiefly (*Nawrocki*) in the medulla, but exist also (*Luchsinger*) throughout the whole length of the cord. The "sweat nerves" reach the nerve-trunks through the sympathetic system.

Remak (see Poliomyelitis Anterior Subacuta), by the analysis of certain facts in pathology, has sought to establish the supposition that there exist groups of ganglion cells in the cord, which control groups of muscles which act functionally together, but which receive their innervation from different nerve-trunks.

Couty (*Gaz. méd. de Paris*, 1876, No. 22) found that section of the posterior spinal roots in frogs had no noticeable effect on the *nutrition* of corresponding parts. He makes the spinal ganglia responsible for trophic changes.

GENERAL THERAPEUTICS OF DISEASES OF THE SPINAL CORD.

BIBLIOGRAPHY.

NUSSBAUM: Nervendehnung bei centralen Leiden. Bayr. ärztl. Intelligenzblatt, 1876, Nr. 8.—VOGT: Die Nervendehnung als Operation in der chirurg. Praxis. Leipzig, 1877. -- EDLEFSEN: Zur Behandlung des Blasenkatarrhs. Deutsch. Arch. f. klin. Med., XIX., 82.

In a case of traumatic paraplegia, in which there occurred strong tonic convulsions, *Nussbaum* stretched the crural and sciatic nerves with the effect of stopping the convulsions, but not influencing the paralysis. The seat of the lesion, whether in cord or in cauda equina, was questionable. *Vogt* comes to the conclusion that nerve-stretching has no effect on central lesions. It is indicated only when there is functional disturbance of a peripheral nerve, either from increased irritability or from disordered circulation. In traumatic tetanus the results are favorable. *Edlefsen* recommends for *catarrh of the bladder*, oil of turpentine (10–12 drops 4 or 5 times a day) and balsam of copaiba. He claims good results from the use of chloride of potassium (solution 1–20 of water, a tablespoonful to be taken every two or three hours).

HYPERÆMIA OF THE SPINAL CORD AND ITS MEMBRANES.

BIBLIOGRAPHY.

Fabre: Des phénomènes spinaux dans les affections cardiques. Gaz. des hôp., 1876, No. 147.

In the last stages of diseases of the heart, *Fabre* has observed pain, anæsthesia, paræsthesia, and slight paresis, rarely also convulsive phenomena, which are probably due to passive hyperæmia of the cord.

APOPLEXY OF THE SPINAL MENINGES.

BIBLIOGRAPHY.

Weber: Spinal Meningeal Hemorrhage. Boston Med. and Surg. J., July 8th, 1875.—**Dowse**: Subarachnoid Hemorrhage of spinal Cord. Trans. Path. Soc., XXVII., p. 1, 1876.—**Lutkenmüller, J.**: Ein Fall von Hæmatorrhachis. Wien. med. Bl., 1878, I., 964, 1879, II., 5.—**Dixon, E. L.**: Intermeningeal Spinal Hæmorrhage Simulating Strychnia Poisoning. Lancet, London, 1879, I., 333. — **Lancereaux**: Hématome de l'arachnoïde comprimant la moité gauche de la moëlle épinière, etc. Rev. méd. franç. et étrang., Paris, 1879, I., 22–25.

Of these records of cases, some of which the writer has not had access to, that of *Dixon* deserves mention. The patient was seized with violent tetanoid convulsions recurring at short intervals, and brought on by any movement. Consciousness was not affected. Death occurred in two hours. On autopsy, the spinal arachnoid cavity was found filled with blood, all other organs being healthy. The resemblance of this case to one of *strychnia poisoning* is its chief point of interest. No chemical examination of the stomach and contents was made. There was no reason to suspect poisoning.

PACHYMENINGITIS AND PERIPACHYMENINGITIS SPINALIS.

BIBLIOGRAPHY.

Pietrulla: Die Pachymening. cervical. hypertrophica. Diss. Breslau, 1876.—Joffroy, A.: Considérat. et observ. relatives à la pachymening. cervic. hypertroph. Arch. génér., 1876, Nov., p. 542.—Lewitsky: Fall von Peripachymening. spinalis. Berl. klin. Woch., 1877, Nr. 17.—Leyden: Fall von Rückenmarkserschütterung durch Eisenbahnunfall. Arch. f. Psych., VIII., S. 31, 1877.—Berger, M.: Zur Kenntniss der Pachymeningitis spinalis hypertrophica. Deutsche medic. Wchnschr., Nos. 50, 51, 53, 1878. Gibney, V. P.: Cervical Pachymeningitis. Med. Rec., N. Y., 1879, XX., 20–22.—Glynn: Internal Hypertrophic Pachymeningitis of the Cord. Brit. Med. J., 1878, II., 805.—Spencer, W. H.: Case of Idiopathic Inflammation of the Spinal Dura Mater. Lancet, 1879, I., 836.—Kohts: Gerhardt's Handbuch f. Kinderheilk., Bd. V.

With reference to the etiology and pathology of these diseases, the following cases are of interest:

The possibility of the occurrence of peripachymeningitis from trauma is indicated by cases reported by *Lewitzky* and *Leyden*. The case reported by the former, *Erb* regards as inconclusive. That of *Leyden* resulted from a railroad accident. The autopsy disclosed caries of the vertebra as its probable point of departure.

Spencer reports a case of suppurative peripachymeningitis apparently primary, and resulting from exposure to cold. Pain in the back and lower extremities without paresis was the main symptom. *Koht's* case was one of *tubercular tumors* on the outer surface of the dura mater in a child. The process seemed to originate in necrosis of a rib with (secondary ?) involvement of the lung. The pressure of the tumor caused degenerative changes in the cord.

Ollivier's case was one of deposit of urates on the anterior surface of the dura mater, and on the sheaths of the nerves in a case of severe chronic *gout*. The symptoms during life, which were probably dependent on this lesion, were a sense of constriction about the neck, thorax, and abdomen, with fulgurant pains in the limbs.

Glynn reports a case of typical cervical pachymeningitis resulting in death in twenty months. Transverse myelitis existed at site of the lesion of the dura mater.

From a clinical point of view, the cases of *Berger* and *Gibney* are of interest. Their cases recovered completely or almost completely after a tedious course of over a year. Cold was the apparent cause in *Berger's* case. Counter-irritation, galvanism, iodide of potassium, and frictions with alcoholic liniments were used. One of *Gibney's* cases, apparently traumatic in its origin (*Med. Rec.*, N. Y., 1880, Sept. 25th), recovered without systematic treatment. Ergot and the iodide of potassium seem to have had some influence over the disease in his experience.

Joffroy calls attention to this relatively favorable course of cervical pachymeningitis as contrasted with that of transverse myelitis.

LEPTOMENINGITIS SPINALIS.

BIBLIOGRAPHY.

SCHWARZ: Méningite spin. Arch. méd. belge, Dec., 1874.—LASÈGUE: Méningite spinale, supposée de nature rhumatism. Arch. génér. de méd., Juin, 1874, p. 743.—SCHULTZE, F.: Das Verhalten des R.-M. und der Rückenmarksnervenwurzeln bei acuter Basilarmeningitis. Berl. klin. Woch., 1876, Nr. 1.—Beitr. z. Pathol. u. pathol. Anat. des centr. Nervensyst. Virch. Arch., Bd. 68, 1876.—VULPIAN: Leçons sur les malad. du systême nerveux, 1877, p. 111.—Meningo-myélite subaiguë. Chir. méd. de l'hôp. de la charité, Paris, 1879, 626–632.—DUNLAP: Meningitis Caused by Penetration of a Fishbone into the Spinal Canal. Brit. M. J., London, 1879, I., 289.

The predominance of the exudation in acute meningitis upon the posterior surface of the cord seems to be best accounted for by the numerous septa which exist in the subarachnoidal space and the consequent greater vascularity of the posterior meninges. This is more satisfactory than the previous supposition that it resulted solely from the position of the patient or (*Vulpian*) from the richer nerve-supply of the posterior meninges.

The case reported by *Dunlap* is of interest because of its unusual etiology. It was one of inflammation of the upper part of the spinal meninges and of those covering the under surface of the cerebrum and medulla, caused by a fishbone which penetrated from the pharynx partly through the second right inter-vertebral foramen and partly through the first inter-vertebral cartilage into the spinal dura mater.

Vulpian states that he has seen rapid improvement in a case of acute spinal meningitis, caused by cold, follow the administration of large doses of salicylate of soda.

TUMORS OF THE SPINAL MENINGES.

BIBLIOGRAPHY.

Dowse: Fibro-nucleated Tumor from the Dura Mater of the Cervical Portion of the Spinal Cord. Brit. Med. J., 1875, May 29th.—Biot: Note sur un cas de sarcome intrarhachid. Lyon méd., 1875, Nr. 31.—Pel: Myxom der Spinalmeningen. Berl. klin. Wchnschr., 1876, Nr. 32.—Gowers: Myo-lipoma of the Spinal Cord. Transact. of the Pathol. Soc., XXXII., p. 19, 1876.—Hünicken: Sarcomatöse Geschwulst im Wirbelcanal. Berl. klin. Wchnschr., 1878, 382.—Bulteau: Tumeur sarcomateuse de la moëlle. Progrès méd., p. 181, 1878.—Wood, H. S.: A Case of Spinal Hydatids. Austral. M. J., 1869, I., 222.

Of these newly reported cases little is to be said. *Gowers'* case was one of *lipoma* containing a large number of transversely striated muscular fibres situated at the conus medullaris; *Wood's,* one of *hydatid cysts* of the lower end of the dura mater, causing it to project out through some of the sacral foramina, hydatids being found also in the liver. In the case reported by *Bulteau,* a spindle-celled *sarcoma,* about the size of the first phalanx of the thumb, was found growing from the pia mater at the junction of the cord and the medulla. Nothing in the clinical histories of these cases calls for special mention.

ANÆMIA OF THE SPINAL CORD.

BIBLIOGRAPHY.

Lauenstein, C.: Zwei Fälle von Embolie der Aorta. Deutsch. Arch. f. klin. Med., XVII., S. 242 u. 491, 1876.—Malbranc, M.: Beob. über Aortenthrombose u. Aphasie.—*Ibid.*, XVIII., S. 462, 1876.—Vulpian: Leçons sur les mal., etc., p. 98, 1877.—Friederich, A.: Recidivirende vorübergehende Rückenmarkslähmung. Virchow and Hirsch Jahresbericht, II., 1879, S. 113.

Under this subject the case related by *Friederich* alone deserves special mention.

The patient was a student, twenty years old, who, since he was eleven or twelve years old, had suffered from repeated attacks of paralysis, the first one following an exposure to cold. In the attack in which *Friederich* saw him, all four extremities became almost completely paralyzed in the course of a few hours without loss of sensation or of reflex action. In two days recovery was complete without treatment. The patient had an hypertrophied heart and a systolic murmur. *Friederich* suggests that a temporary anæmia of the cord was the cause of the attacks.

SPINAL APOPLEXY.

BIBLIOGRAPHY.

WEBBER: Fall von Spinalhämorrhagie. Schmidt's Jahrb., Bd. 170, S. 25, 1876.—FOX, E. L.: On Spinal Hemorrhages. Med. Times and Gaz., 1876, Aug. 23d.—REMAK, E.: Fall von atroph. Spinallähmung durch traumat. halbseit. Blutung in die Halsanschw. des R.-M. Berl. klin. Woch., 1877, Nr. 44.—VULPIAN: Leçons, p. 92, 1877.

Beyond the bibliography, the case reported by *Remak* alone will be alluded to. This was a case of atrophic paralysis on one side of the body, resulting from an injury which *Remak* supposed to be a hemorrhage confined to one-half of the cord in the cervical region.

ACUTE TRAUMATIC LESIONS OF THE SPINAL CORD.

BIBLIOGRAPHY.

FEINBERG: Wirbelfractur und Rückenmarksabscess. Berl. klin. Woch., 1876, Nr. 32.—HULKE: Three Cases of Broken Neck. Med. Times, 1876, July 29th.—HAYEM: Arthrite de l'articulation de l'axis avec l'atlas, etc. Gaz. des hôp., 1876, No. 147, Soc. d. Biol.—SEELIGMÜLLER: Fall von geheilt. Fractur d. Lendenwirbel. Deut. med. Wchnschr., 1877, Nr. 28.—HEYNOLD, H.: Fall von Luxation und Fractur des 6. u. 7. Halswirbels, verbunden mit ungewöhnl. nied. Temperaturabfall. Berl. klin. Woch., 1877, Nr. 39.—NIEDEN, A.: Ueber Temperaturveränderungen (Hyperpyrexie und Apyrexie), bedingt durch Verletzungen des Halsrückenmarkes. Berl. klin. Wchnschr., No. 50, 1878.—RIGLER, JOHS: Ueber die Folgen der Verletzungen auf Eisenbahnen, insbesondere der Verletzungen der Wirbelsäulen und des Rückenmarks, 1879.—BECK. B.: Ueber Verletzungen der Wirbelsäulen und des Rückenmarks. Arch. f. path. Anat., etc., Berl., 1879, 207-255.

Injuries to the spinal cord, without lesion of the spinal canal or of the soft parts, occur more readily when there already exists some anomaly of the spinal column, as in the case of *Hayem*, in which there existed hypertrophy of the odontoid process.

Beck records a case of softening of the cord in the dorsal region with a clot between the dura mater and the bone, which was traumatic in its origin, but without fracture of the vertebræ.

LANE LIBRARY. STANFORD UNIVERSITY

GRADUAL COMPRESSION OF THE SPINAL CORD.

BIBLIOGRAPHY.

Westphal: Arch. f. Psych., II., S. 374, 1870.—**Frommann**: Fall von Wirbelcaries u. Degener. des R.-M. Virch. Arch., Bd. 54, 1872.—**Pierret**: Plusieurs cas de névrite parenchym., etc. Arch. de physiol. norm. et path., VI., 1874, p. 968.—**Gowers**: Caries of Dorsal Spine, etc. Med. Times and Gaz., 1876, Nov. 4th.—**Ramskill**: *Ibid.*, Nov. 18th.—**Kadner**: Zur Casuistik der Rückenmarkscompression. Arch. de Heilk , XVII., 1876, S. 481.—**Hayem**: Gaz. des hôp., 1886, Nr. 147.—**Couty**: Note sur les troubles vasomot. et therm. observés dans un cas de compress. d. l. moëlle. Gaz. médic., 1876, No. 37.—**Vulpian**: Leçons, l. c., p. 14, 1877.—**Masse**: De la compression lente de la moëlle épinière. Montpel., 1879.

The secondary degeneration which, in the cases of *Westphal, Frommann,* and *Kadner,* was observed to extend upward for a short distance above the point at which the cord was compressed at the periphery of the cord, was evidently situated in the cerebellar lateral column tracts of *Flechsig.*

In regard to *vasomotor disturbances* in these cases, *Vulpian* speaks of various varieties as occurring from irritation and paralysis of vaso-dilator and vaso-constrictor fibres.

Although, as a rule, *motor disturbances* show themselves first in compression of the cord from Pott's disease, yet, as in *Ramskill's* case, disturbance of sensation may be first in order of time. On the other hand, compression from behind may cause predominant disturbance of motion, according to *Vulpian,* because the gray matter suffers less from pressure than the white.

Although, as a rule, there is increase of reflex action in these cases, *Kadner* has shown that in many this increase may not be present.

CONCUSSION OF THE SPINAL CORD.

BIBLIOGRAPHY.

Willigk, A.: Anatom. Befund nach Hirnerschütterung. Prag. Vierteljahrschr., Bd. 128, S. 19, 1875.—**Bernhardt**, M.: Ueber die Folgen der Gehirns- und Rückenmarkserschütterung nach Eisenbahnunfällen. Berl. klin. Wchnschr., 1876, Nr. 20.—**Buzzard**, T.: A Railway Case; Shock from an Unexpected Descent, etc. Transact. of Clin. Soc., London, Vol. IX., 1876.—**Leyden**: Fall von Rückenmarkserschütterung durch Eisenbahnunfall. Arch. f. Psych. u. Nerv., VIII., S. 31, 1877. —**Eberhard**, **Gust.**: Ueber die Erschütterung des Rückenmarks

Götting., 1878.—BERNADIS, J. B. E.: De la commotion de la moëlle épinière. Montpel., 1879.

In a case reported by *Leyden* as concussion of the spinal cord, peripachymeningitis with cheesy tubercular meningitis and secondary myelitis from compression were found to have developed. *Willigk,* in a case of concussion which died three months after the accident, claims to have found the capillaries and the small arteries and veins throughout both cord and brain widely dilated and showing slight fatty degeneration of their walls.

Buzzard has observed disturbance of speech and of the functions of the tongue with glycosuria as the result of concussion.

Bernhardt and *Leyden* have added to the list of cases in which the morbid symptoms appeared after a considerable time had elapsed since the injury. *Erb* has also seen several such cases.

SPINAL IRRITATION.

BIBLIOGRAPHY.

COGHILL, J. G. S.: Irritable Spine as an Idiopathic Affection. Brit. Med. J., London, 1879, II., 571–578.

Coghill reports four cases of irritable spine as treated successfully by tonics and by the local application of *Corrigan's bouton,* heated to a blue heat, over the painful spots.

The cases reported by *Benedickt* (Neue Behandlungsmethoden der Spinal-Irritation. *Wien. Med. Presse,* 1879, 105, 173) are cases of pure hysteria, not of spinal irritation in *Erb's* sense.

NEURASTHENIA SPINALIS.

BIBLIOGRAPHY.

HOLST: Ueb. Neurasthenie und üb. ihr Verhältniss zur Hysterie und Anæmie. Dorp. med. Zeitschr., VI., S. 15, 1876.—ANJEL: Ueber vasomotor. Neurasthenie, etc. Arch. f. Psych. u. Nerv., VIII., S. 394, 1878.

Erb protests against the classification of this disease as a manifestation of *hypochondria* (*Jolly*). Though often combined with the latter, it is to be regarded as of distinctly spinal origin. The recent literature given above calls for no comment.

MYELITIS.

BIBLIOGRAPHY.

v. d. Velden u. Leyden: Fall von acuter aufsteig. spinaler Paralyse. Deutsch. Arch. f. klin. Med., XIX., S. 333, 1877.—Lauinger, C.: Beitr. z. Lehre von d. acuten Myelitis. *Ibid.*, XIX., S. 424, 1877.—Vulpian: Leçons sur les mal. des syst. nerv., p. 144, 1877.—Leyden: Ueber experiment. erzeugte Rückenmarkssklerose u. die Ausgänge der acut. Myelitis. Berl. klin. Woch., 1877, Nr. 49.—Charité-Annal., III. (1876), S. 248, 1878.—Grasset: Mal. du syst. nerveux, 1878.—Westphal: Ueb. combinirte (primäre) Erkrankungen der Rückenmarksstränge. Arch. f. Psych., VIII., S. 469, 1878.—Kahler und Pick: Ueb. combinirte Systemerkrankungen des R.-M. *Ibid.*, VIII., S. 251, 1878.—Prout et Joffroy: Contribution à l'étude de la myélite aiguë. Observ. de myélite à début apoplectiforme, etc. Cong. périod. internat. d. sc. méd. Compte-rendu, Genève, 1878, 157–159.—Caizergues, R.: Thérapeutique de la myélite syphilitique. Rev. méd. franç. et étrang., Paris, 1878, II., 353, 385, 417.—Schuster: Ein Fall von acuter Myelitis. Berl. klin. Wchnschr., 1879, XVI., 292.—Atkinson: On the Morbid Histology of the Spinal Cord in Five Cases of Insanity.—Leyden, E.: Beiträge zur acuten und chronischen Myelitis. Ztschr. f. klin. Med., Berl., 1879, I., 1–26.—Ueber die durch plötzliche Verminderung des Barometerdrucks entstehende Rückenmarksaffection. Arch. f. Psychiat., 1879, IX., 316–324.—Babesiu, V.: Ueber die selbstständige combinirte Seiten- und Hinterstrangsclerose des Rückenmarks. Virch. Arch., 1879, Bd. 76, S. 74–84.—Vallin, E.: Note sur quelques formes du rhumatisme spinal. Gaz. des hôp., No. 18, 1878, p. 188.

Vallin reports three cases of more or less complete paralysis of the extremities, two being associated with disturbances of sensation, occurring in connection with *acute articular rheumatism.* In two, the symptoms rapidly passed away, a slight hemiplegia remaining in a third two months after the inception of the disease, at which time the patient passed from under observation. *Erb* considers it doubtful whether arsenic, phosphorus, mercury, bisulphide of carbon, alcohol, or lead can give rise to acute myelitis.

Leyden reports three cases of a peculiar form of myelitis resulting from *sudden diminution* of *barometric pressure.* One case was that of a laborer who, half an hour after coming out of a caisson, where he had been working under increased atmospheric pressure, became suddenly paraplegic; death occurred in the fifteenth day, the patient having the usual symptoms of acute myelitis with grave vesical symptoms. The cervical and lumbar parts of the cord were healthy. In the dorsal portion were seen, scattered through the cord, particularly in the posterior columns and in the posterior parts of the lateral columns, accumulations of large, round, nucleated cells which pushed apart the nerve-fibres, and contained among them only a few normal vessels—no neuroglia, no blood pigment. The nerve-fibres in their neighborhood showed in greater or less extent signs of parenchymatous myelitis. The gray matter was normal. *Leyden* believes that the process consisted in a splitting of the masses of nerve-fibres by oxygen or carbonic acid set free from the capillaries without rupture of their coats or with rupture of only such small vessels that no noticeable hemorrhage took place, and in the subsequent filling up of these spaces with cells. The dorsal portion of the cord was probably affected because of its normally lesser consistency than the enlargements. The other two cases were similar, except that one patient was discharged completely recovered in nineteen days, the other recovered incompletely in thirty days, at which time he was discharged from the hospital.

Hayem, by tearing out the sciatic nerve of young rabbits, caused a cicatricial myelitis which became the point of departure of a progressive central myelitis with muscular atrophy. The process consisted in a degenerative atrophy of the ganglion cells. A similar, though slower result followed simple section of the sciatic nerve.

Leyden's latest experiments in the artificial excitation of myelitis have enabled him to demonstrate the passing over of acute myelitis into cyst-formation and sclerosis. *Vulpian* has observed similar processes after injecting nitrate of silver into the cord.

v. d. Velden described a case in which numerous small foci of acute myelitis were found in the upper thoracic and cervical portion of the cord predominantly in the anterior and lateral columns and in the anterior horns. The process was characterized by changes in the nerve-fibres, slight exudation around the vessels, and slight increase in the number of glyoma cells. *Lauinger, C.*, has published a case in which the process was even more purely parenchymatous. These two cases followed the clinical course of acute ascending paralysis. In that of *v. d. Velden*, the galvanic and faradic irritability was fully lost on the second day of the illness, while in that of *Lauinger* the faradic irritability was fully retained. The reflexes in the latter, which at first were fully lost, returned in a few days; an apparent exemplification of the *inhibitory effect* of the acute affections of the cord suggested by *Goltz*. In the case reported by *Leyden* (see above), faradic irritability of the muscles was good three days after the beginning of the disease.

A case reported by *Schuster* of acute myelitis is of interest on account of its probable syphilitic origin, and the good, although not complete, recovery after five months of treatment by warm baths, electricity and *mercurial inunctions.*

CHRONIC MYELITIS.

Since the discovery by *Flechsig* of the various tracts or systems in the cord, of which an account is given above, several observers have endeavored to discover the relations which exist between these systems and certain previously classified diseases of the cord (lateral sclerosis, amyotrophic lateral sclerosis, etc.), and have also endeavored to differentiate a new class of diseases which is characterized by a primary affection of a number of these systems to the exclusion of others. This latter class, the *combined-system diseases* of the cord, will be here spoken of. The relation between *Flechsig's* discovery and the previously known diseases will be alluded to under the appropriate headings.

Kahler and *Pick* have described a case in which at least four systems were involved, and in which the degeneration confined itself approximately within their limits. Then there are cases in which the myelitic process is not confined strictly within the limits of any distinct systems, but is spread, more or less widely, over the section of the cord, as in cases described by *Westphal*, "in which there can be no question of a limitation of the affection to the systems of *Flechsig*. Such cases are very frequent and seem not unfrequently as if they were the result of a disease which began as a system disease, but which came to be examined at a later stage" (*Erb*).

The most important recent contributions to this subject have been from *Babesiu*, *Leyden*, and *Westphal*.

The case reported by *Babesiu* clinically presented all the symptoms of spastic paralysis, with the addition of atrophy of the optic discs, some loss of sensation in the feet, retention followed by incontinence of urine, and dragging pains in the feet and genitals. On autopsy, in addition to some lesions of the brain, etc., which had no probable connection with the symptoms observed in the limbs during life, degeneration of the lateral columns was found, most marked in the thoracic portion, where the degeneration reached from the posterior almost to the anterior horns. In the cervical expansion, the degeneration of the lateral columns dwindles down to a small triangle at their periphery, being separated from the posterior horns by healthy tissue. Downwards a similar diminution takes place. Only in the periphery of the cord does the degeneration approach the anterior columns. Wedge-shaped, sunken-in, sclerotic spots were found in the lateral cerebral tracts above the anterior pyramid. The posterior columns in their posterior periphery, and the columns of Goll were tolerably evenly affected throughout the cord. The central canal was filled throughout with growing cells. Ganglion cells of the anterior horns and Clark's columns normal. The pia mater of the cord was thickened, especially in its posterior and lateral parts, and was also congested and strongly pigmented. This short abstract of the case gives an idea of the class of cases which are grouped under the heading "combined-system diseases of the cord." It is unnecessary to state how manifold the symptoms may be in these cases, according to the particular system affected, and how still more confused may be the symptom-complex when the disease is of the irregular form.

Leyden believes that only two forms of primary "system diseases" of the cord have been demonstrated, *i. e.*, tabes dorsalis and atrophy of the motor parts of the cord (degeneration of the cells of the anterior horns, motor tracts, spinal nerve-roots, and nerves of the muscles). A combination of these two may exist, giving rise to a "combined-system disease." *Kahler* and *Pick*, *Westphal* and *Babesiu*, he thinks, have systematized their cases more definitely than the clinical facts warrant. He cites several cases of his own, one of which resembled very closely that reported by *Kahler* and *Pick*, and states that he believes that all the cases of this class are cases of simple extension of disease by continuity of tissue with typical ascending and descending degeneration.

Westphal (Ueber combinirte (primäre) Erkrankung der Rückenmarksstränge, *Arch. f. Psych.*, Bd. IX., 691–737) analyzes his previously reported cases. He concludes that, although the paralysis in these cases bore no constant relation to the lesions of the lateral columns, yet this was the main element in its causation. The degeneration of the muscles was also an element. Implication of the lateral columns without paralysis, as in *Friedreich-Schultze's* case (Virch. *Arch.*, Bd. 70, S. 141), might have been due to non-destruction of the conducting fibres by the morbid process. Absence of muscular rigidity in his own cases, in which both lateral and posterior columns were involved, he accounts for by the extension of the affection of the posterior columns in the lumbar region to the posterior root-zones, by which extension reflexes were prevented. The supposition that the inflammatory process was transmitted by continuity of tissue or through the pia mater he rejects, because in all his cases healthy tissue intervened between the posterior and the affected portions of the lateral columns, and the pia mater was thickened only over the posterior surface of the cord. In one case, the affected portion of the lateral columns did not extend to the periphery. *Westphal* concludes that in certain cases of myelitis the process may extend in the long axis of the cord, at least in the lateral and anterior columns, and, in general, symmetrically; that there is no manifest single point of departure of the process; and that there are no *pure* system diseases, although here and there are indications that this or that system may be affected as such. Some undiscovered changes in the gray matter of the cord, which was apparently normal in Westphal's cases, may underlie these cases. The existence of some change is suggested by the *red* reaction of otherwise normal appearing ganglion cells, in diseased cords, with methyl-violet (*Jürgens*), whereas in normal cords the ganglion cells color *blue* with this reagent. *Westphal* rejects the idea that what in one of his cases looked like typical ascending and descending *secondary* degeneration (and is so regarded by *Leyden*), was so in fact, because the portion of the cord which would have to be assumed as that primarily affected was not involved in its whole diameter, and in cases of disseminated sclerosis the isolated foci of disease have never been observed to be the points of departure of secondary degeneration. He therefore concludes that the affection of the columns of Goll, the cerebellar-lateral tracts, and the lateral pyramid tracts observed in this case were *primary* degenerations of these systems.

Atkins found irregularly disseminated myelitis in three cases of *in-*

sanity of different types. In one case of general paresis extensive softening of the lumbar and sacral regions of the cord was found.

Erb quotes the following *principles* from *Renz*, of Wildbad, which should control the employment of *thermal baths* in myelitis: During the active stages of disease, harm is apt to be done unless the greatest care is exercised in their use. This rule holds good, whether the disease is acute or chronic. The baths must be cooler, less frequent, and of shorter duration the more obviously progressive a chronic disease is. Acute cases should not use them till they are convalescent. When a case is improving, the time and temperature of the baths may be increased cautiously. Only those cases in which the meninges are prominently affected endure baths of high temperature and long duration with benefit.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

BIBLIOGRAPHY.

Charcot: Diagnostic des formes frustes de la sclérose en plaques. Progrès méd., Paris, 1879, VII., 97-99.—Frommann, C.: Untersuchungen über die Gewebsveränderungen bei der multiplen Sklerose des Gehirns und Rückenmarks. Jena, 1878.—Pitres, A.: Contribut. à l'étude des anomalies de la sclérose en plaques disséminées. Rev. mensuelle, Dec., 1877, p. 893.—Da Costa: Multiple spinal Sclerosis (Case). N. Y. Med. Rec., 1879, XV., 313.—Westphal, C.: Ueber strangförmige Degeneration der Hinterstränge mit gleichzeitiger fleckweiser Degeneration des Rückenmarks. Arch. f. Psych., 1879, IX., 389.—Pollard, F.: Case of Disseminated Cerebro-spinal Sclerosis in a Child. Lancet, Aug. 10th, 1879, p. 183.—Dickenson, Cheadle, and Dreschfeld: Cases of Disseminated Sclerosis in Children. Med. Times and Gaz., 1879, I., pp. 112, 139.—Bristow: A Case of Disseminated Sclerosis. Med. Times and Gaz., 1879, I., 673.—Lomikowsky, M.: Laryngoscopischer Befund bei Sclerose en plaques cérébrospinales. Berl. klin. Wchnschr., 1879, 610.—Pollak, L.: Multiple Herdsklerose des Hirns und Rückenmarkes im Säuglingsalter. Deutsch. Arch. f. k. Med., 1879, XXIV., 407-415.—Leyden, E.: Beiträge zur acuten und chronischen Myelitis. Ztschr. f. klin. Med., Berl., 1879, I., 1-26.

This abundant literature furnishes little new information.

The number of cases (all without autopsies) reported in children from five months to eight years of age is somewhat remarkable (*Dickenson. Pollak*).

Two of *Dreschfeld's* cases were brothers.

Westphal's case was one which presented the irregular complex of symptoms characteristic of multiple spinal sclerosis. The autopsy disclosed columnar degeneration of the posterior columns, with multiple sclerosis of the lateral columns.

Leyden reports a case in which all the symptoms of the disease appeared, but in which almost complete recovery (slight difficulty in speaking, tremor and weakness of legs remaining) occurred after treatment by galvanism and a bath-cure at Rehme. This case he regards as one of acute, multiple cerebro-spinal inflammation which resolved and did not go on to sclerosis. He thinks that the case tends to prove that sclerosis is a late stage of an acute process, not a distinct process in itself.

LOCOMOTOR ATAXIA.

BIBLIOGRAPHY.

Bush: Ein Fall von Tabes dorsalis; Schwinden der Sehnenreflexe nach mehrjähriger Dauer der Krankheit. Petersburger med. Wochenschr., No. 46, 1878.—Kahler: Casuistischer Beitrag zur Therapie der typischen Tabes. Prager med. Wochenschr., No. 36, 1878.—Briersion: Contribution à l'étude du tabes dorsalis ataxique. Thèse, Paris.—Onimus: De la contracture dans l'ataxie. Gaz. des hôp., No. 88, 1878.—Audibert: Des phénomènes viscereaux dans l'ataxie locomotrice progressive. Marseille méd., 1878, XX., 521–530.—Kahler, O., und Pick, A.: Beitrag zur Pathologie und pathol. Anat. des Centralnervensystem. Vrtljschr. f. prakt. Heilk., Prag, 1879, I., 1–86.—Erlemeyer, A.: Ueber Tabes dorsalis incipiens. Cen. Bl. f. schweiz. Aerzte, Basel, 1879, IX., 3–7. - Ferry, Jules: Recherches stastistiques sur l'étiologie de l'ataxie locomotrice progressive. Par., 1879.—Beluger, A.: Note sur le traitement de l'ataxie locomotrice par les eaux de la Malon. Ann. Soc. d'hydrol. méd. de Paris, 1879, III., V., 3. —Petit, L. H.: De la ataxie locomotrice dans ses rapports avec le traumatisme. Rev. mens. de méd. et de chir., Paris, 1879, III., 209–224.—Berger: Zur Symptomatologie der Tabes dorsualis; d. schles. Gesellsch. f. vaterl. Cultur. Bresl., 1878, IV., 216.—Friedreich: Nystagmus bei Ataxie. Bericht über die Versammlung der ophthalmolog. Gesellschaft, 198.—Kahler und Pick: Zur Lehre von der Ataxie. Vrtljsch. f. d. prakt. Heilk., Prag., 1879, II., 86–97.—Robert, A.: Ataxie locomotrice; amyotrophie secondaire des membres supérieurs; arthropathie du coude gauche. Progrès méd., Par., 1879, VII., 326.—Berger: Zur Aetiologie der Tabes dorsalis. Breslau ärztl. Ztschr., 1879, I., 70.—Vulpian: Ataxie locomotrice progressive (5 cases). Chir. méd. de l'hòp. de la Charité. Par., 1879, 807–832, 823–828.—Erb, W.: Zur Pathologie der Tabes dorsalis. Deutsches Arch. f. klin. Med., 1879, XXIV., 1–52.—Christian, J.: Des rapports de l'ataxie locomotrice progressive avec la paralysie générale. Union méd., 1879, XXVIII., 157.—Hardy: Ataxie locomotrice. Gaz. des hôp., Paris, 1879, LII., 1033.—Vallin: Des alterations trophiques des os maxillaires dans l'ataxie locomotrice. Union méd., Par., 1879, XXVIII., 737–749.—Drosdoff: Archiv f. Psych., IX., 203, 1879.—Berger: Ueber Sehnenreflexe. Centrabl. f. Nervenheilk. und Psych., etc., 1879, No. 4.

With reference to the *etiology* of tabes dorsalis it is to be noted that *Vulpian* affirms that *hysteria*, especially in its convulsive form, has a decided influence on the development of the disease. Among forty-four of the cases analyzed by *Erb* in a recent article, twenty-seven were found to have had *syphilis*, which usually had developed several years before the tabes, and which was usually light in its antecedent symptoms. *Fournier* found twenty-four out of thirty, and *Vulpian* fifteen out of twenty cases of tabes which were syphilitic. *Erb* is therefore inclined to believe that syphilis has a more direct connection with tabes than he has formerly held.

Kahler and *Pick* advance the supposition that ataxia and other forms of spinal disease, occurring after acute infectious diseases, are the result of accumulations of the fungi which cause the acute diseases, in the central nervous system, and which give rise to nutritive disturbances of greater or less gravity and extent.

As examples of the previously known fact that the morbid process in tabes is in most, perhaps in all cases not confined to the posterior columns, cases reported by *Hayem*, *Prévost*, *Kahler* and *Pick*, and *Westphal* may be cited. In *Hayem's* case, sclerosis of part of the lateral columns, numerous changes in the gray substance, sclerosis of the ascending root of the trigeminus and of the so-called "respiratory bundle" existed in addition to a lesion of the posterior columns. The cases of *Westphal* and *Kahler* and *Pick* have been referred to under *myelitis* as cases of combined system-diseases of the cord. Also a case communicated by *Friedreich* (*Virch. Arch.*, Bd. 70) is to be mentioned, in which an extensive annular degeneration existed, resembling the lesion found in one of *Kahler* and *Pick's* cases. In all these cases, the *direct cerebellar lateral tracts* were involved in addition to the posterior columns. The supposition is advanced that the ataxia results from the implication of those columns and not from the disease of the posterior columns. This supposition is far from being proved.

In his recently published analysis of fifty-six clinical cases of locomotor ataxia occurring in his own practice, *Erb* gives a tabulated statement of the relative frequency and importance of the symptoms of the disease which cannot be transcribed here in full, on account of my limited space. In this paper he lays greater stress than formerly on the occurrence of *spinal myosis*, or, as he proposes to call it, *reflex immobility of the pupil*, to distinguish the condition from immobility of the pupil during accommodative effort and from complete immobility, and to include those cases in which the pupil fails to respond to light, but in which it is of normal size or is dilated. *Erb* found the symptom in fifty-four per cent of patients examined. *Vincent* found it in ninety-two per cent. It is probably not an early symptom. It is only found in progressive paralysis of the insane with equal frequency (nineteen out of twenty-one cases *Vincent*). *Atrophy of the optic nerve* occurred in only six out of forty-nine cases. *Tendon reflex* was absent in all but one of fifty cases. This is *probably* always an early symptom. Mechanical irritability of the quadriceps was always fully preserved (tested in thirty-two cases). *Erb* has found tendon reflex absent in only two other morbid conditions, *i. e.*, progressive cerebral paralysis and in cases of paresis of the quadriceps with atrophy and degeneration reaction. In *Erb's* experience, it is never absent in young or middle-aged people in health. Hence he thinks that its absence may be regarded as almost pathognomonic of tabes in its earlier diagnosis. *Cutaneous reflex* was absent in six out of forty-seven cases. *Analgesia* to *strong* irritant (*Berger*) was present in thirteen out of twenty-nine cases. This symptom, which is not rare in other diseases, is often early present, but it must be remembered how widely sensibility varies in normal subjects.

Oulmont, by careful examination of the *disturbance of sensibility* in ataxic subjects, discovered that it was not only very frequent, but very widespread. He discovered certain spots of predilection (in the breasts, around the umbilicus, fingers and forearms, backs of the legs, the heels and toes, etc.).

Experiments to determine the farado-cutaneous sensibility in tabes conducted by *Drosdoff* (seven cases), and *Erb* (four cases), showed diminution all over the body. A broad, soft wire brush was used. It appears to be an early symptom and may prove of diagnostic value (*Erb*).

Remak, E., in two cases discovered that sensibility could be exhausted by an abnormally small amount of irritation.

Pierret has found that all possible nervous *disturbances of hearing* may precede the ataxic symptoms. *Althause* has recorded a case of the same sort.

Among *unusual cases* may be mentioned the following: A case of "hereditary" tabes in which attacks, several hours long, of spasmodic coughing, great feeling of anxiety, rapid respiration, dyspnœa, and cyanosis with frequent and rapid pulse (bronchial crises) is recorded by *Kahler* and *Pick*. The same observers record a case of ataxia following *malarial fever*, with recovery after two months under quinine. Slight nystagmus and slight stiffness of the legs remained. The nystagmus, difficulty of speech, and absence of pains and of sensory disturbances seem to ally this case with that reported by *Leyden* as acute cerebro-spinal sclerosis with cure, rather than with locomotor ataxia. *Erb* records a case of not very typical but distinct ataxia following diphtheria (?) and exposure to cold which recovered rapidly, under galvanic treatment, in six months.

Ataxia associated with lesion of the left olivary body and a unilateral case with a tuberculous nodule in the middle of the left parietal lobe are recorded by *Kahler* and *Pick*. The same observers record a case of ataxia of apparently *cerebellar origin* in which the *tendon reflex was very powerful.* They record also a case of tumor of the third dorsal vertebra in which ataxia preceded all the symptoms of the transverse myelitis which subsequently developed.

Robert records a case of sclerosis of the posterior columns and of the anterior cornua of the brachial enlargement which, in life, was a case of typical tabes associated with marked atrophy of the forearms and hands. *Erb* and *Hardy* record two similar cases of this somewhat rare combination (both clinical).

Kellogg reports two cases very imperfectly, which seem to fall under *Friedreich's* form of tabes, and which developed in brothers at their sixth year. In other branches of the same family were other similar cases. *Kahler* and *Pick* have reported a case in which four systems were involved, viz., the pyramid tracts, the cerebellar lateral tracts, with Clark's columns, the wedge-tracts, and the columns of Goll which, as the affection was associated with defective development of the cord, they have tried to rank as a case of hereditary tabes. As the ataxia was very slight, the paralytic symptoms prominent, and there were no disturbances of the bladder or of sensibility, the justice of this classification seems questionable (*Erb*).

The rules given by *Renz* (see myelitis) in regard to the use of *thermal baths* in myelitis hold good for tabes also. He begins with a bath of 31.0°–32.5° C., of 5–10 minutes' duration. Every two or three baths he decreases the temperature ½° until 29° or 28° is reached. When lancinating pains are present, baths from 33° to 34° of 8–15 minutes' duration are best. *Sée* obtained good results, when the pains were prominent, from salicylate of soda.

Erb recommends the energetic use of the combined antisyphilitic treatment, when syphilis coexists in a case of tabes, much more strongly than formerly. In his limited experience with it, he has seen encouraging results. In one case of atrophy of the optic discs, of two and a half years' standing, he saw material improvement of vision under galvanic treatment.

SPASTIC PARALYSIS.

BIBLIOGRAPHY.

Erb: Ueber das Vorkommen der spastischen Spinallähmung bei kleinen Kindern. Betz' Memorabil., 1877, Heft 12, S. 529.—Seeligmüller: Ueb. spast. spinale Paralysen bei Kindern. Amtl. Bericht üb. d. 50. Vers. deutsch. Naturforscher u. s. w., in München, 1877, S. 299.—Flechsig, P.: Ueber Systemerkrankungen im R.-M. 4. Artikel; Primäre Degeneration der Pyramidenbahnen. Arch. d. Heilk. XIX., S. 53, 1878.—Pick, A.: Zur Lehre von den Systemerkrankungen des R.-M. Fall von Sclerose later. amyotrophique. Arch. f. Psych., VIII., S. 294, 1878.—Stoffella: Ein Fall von Seitenstrangsclerose des R.-M. Wien. med. Wochenschr., 1878, Nr. 21 und 23.—Leyden: Ueber spastische Spinallähmungen. Berl. klin. Wchnschr., 1878, No. 48.—Velden, R.. Fall von spastischer Spinalparalysie. Heilung. Berl. klin. Wchnschr., 1878, No. 38.—Henck, G.: Ein Fall von acuter spastischer Spinalparalysie. Berl. klin. Wchnschr., 1879, XVI., 29–31.—Seguin, E. C.: The Present Aspect of the Question of Tetanoid Paraplegia. Arch. of Med , N. Y., 1879, I., 74-83.—Westphal: Ein Fall von spastischer choreatischer Paralysie. Charité-Ann., Berl., 1879, IV., 421.—Schulz, R.: Gibt es eine primäre Sclerose der Seitenstränge des Rückenmarks? Deut. Arch. f. klin. Med., 1878–9, XXIII., 343–356.—Zunker, E.: Mittheil. v. Krankheitsfällen welche unter den Sympt. der spastischen Spinalparalysie einhergingen. Charité-Ann., Berl., 1879, IV., 241–255.—Nixon, C. J.: Clinical Contributions to Diseases of the Spinal Cord. Dublin J. M. Sc., 1879, LXVII., 289-291.—Feinberg: Fall von spastischer Spinalparalyse. Berl. klin. Wchnschr., 1879, XVI., 290–292.—Charcot: Sclérose latérale amyotrophique, etc. Gaz. de hôp., 1879, LII., 1065–1082.—Ricklin: De la paralysie spinal spasmodique.

Gaz. méd. de Paris, 1878, Nos. 27, 29, and 31.—Shaw, J. C.: Case of Progressive Muscular Atrophy with Sclerosis of Lateral Columns. Jewell's J. of Nerv. and Ment. Dis., Jan., 1879.—Mitchell, S. W.: Spasmodic Disorders of the Legs. N. Y. Med. Rec., June 28th, 1879, p. 601.—Amyotrophic Unilateral Spinal Sclerosis (case). Boston M. and S. J., 1879, C., 248.—Westphal, C.: Ueber combinirte (primäre) Erkrankungen der Rückenmarkstränge. Arch. f. Psych., Bd. VIII., S. 469, and IX., S. 691.—Babesiu, V.: Ueber die selbstständige combinirte Seiten- und Hinterstrang Sclerose des Rückenmarks. Virch. Arch., Bd. 76, S. 74–84.—Minot, F.: Cases of Diseases of the Spinal Cord. Boston M. and S. J., 1879, C., 432.—Seeligmüller, A.: Ueber Lähmungen im Kindesalter. Jahrb. f. Kinderheilk., XII., S. 321, XIII., S. 226–315.—Mader: Einige Fälle seltener Spinalkrankheiten. Wien. med. Presse, 1879, 961, 995, 1058.—Stumpell: Zur Kenntniss der Sehnenreflexe. Deutsch. Arch. f. klin. Med., 1879, XXIV., 175–191.—Gowers, W. R.: A Study of the So-called Tendon Reflex Phenomenon. Proc. Roy. M. and Chir. Soc., London, 1878–79, 366, and a few others.

Seeligmüller calls attention to the possibility of the relationship of parents being an *etiological* factor in spastic paralysis in cases which he has observed among children.

No case has yet been published which proves the existence of a primary disease limited to the pyramid tracts satisfactorily. *Stoffella* has published a typical, uncomplicated case of spastic paralysis, in which, on autopsy, gray degeneration of both lateral columns, principally in their posterior portions and in the lower thoracic and lumbar portions of the cord, was found. In these portions of the cord, the degeneration reached to the meninges externally, and to the posterior horns internally. Unfortunately the absence of any record of the condition of the brain and medulla, and of any microscopical examination of the cord, renders the case incomplete, and therefore inconclusive.

Erb supposes that the lesions actually found in the lateral columns in *amyotrophic lateral sclerosis* represent the type which we may expect to find in an uncomplicated case of spastic paralysis. The degeneration in these cases is found predominantly in the lateral pyramid tracts, with a much less marked lesion in the principal mass of the anterior columns, and the anterior mixed region of the lateral columns. The anterior pyramid tracts are also sometimes degenerated. The degeneration of the pyramid tracts is marked, involving atrophy of the nerve-fibres, etc. In the other tracts of the white matter of the cord, which may be involved, an interstitial growth of the neuroglia is found without marked degeneration or atrophy of the nerve-fibres (*Flechsig* and *Pick*). The pyramid tracts are affected throughout the whole length of the cord, and the lesion has been traced through the pons into the crura cerebri.

The anterior gray columns are degenerated, there being nothing typical of this particular disease in the microscopic appearances. The cells of the tractus intermedio-lateralis, those of Clark's columns, and of the posterior gray columns are normal. The lesion of the gray matter is usually most prominent in the cervical enlargement.

The changes characteristic of bulbar paralysis are found in the medulla. The anterior roots of both cord and medulla are gray, degenerated, and atrophic. The muscles, especially of the upper extremities, present about the same appearance as in typical progressive muscular atrophy.

Flechsig and *Pick* have concluded that, in *amyotrophic lateral sclerosis,* we have to do with a disease affecting the *whole system of nerve-fibres and ganglion-cells, which unite the motor centres in the cortex of the brain with the muscles.* Both these observers are inclined to regard the process as a primary disease of the nervous elements, a so-called parenchymatous degeneration or sclerosis. *Flechsig* seems inclined to regard the lesions of the anterior parts of the lateral columns, and of the principal masses of the anterior columns, as an accidental and immaterial complication. *Pick* regards them as being continued from the anterior roots and the primarily affected pyramid tracts.

With reference to the *symptomatology* of spastic paralysis, *Erb,* in the second edition of his work, calls attention to the great prominence of *contracture* in children affected with the disease. The increase of tendon reflex may usually be found in them if care is exercised in bringing the limbs into a proper position for its demonstration. In testing the ankle-clonus too strong or sudden flexion must be avoided.

Numerous cases of more or less typical spastic paralysis are reported clinically in recent literature. Many autopsies of cases, in which spastic symptoms have been prominent, have also been reported and shed some light on the affection. *Hallopeau* (Des paralysies bulbaires. Thèse, Paris, 1875, p. 121) and *Schulz* each reports a case of tumor of the medulla, with secondary degeneration of the pyramid tracts, in which spastic symptoms were prominent, though associated with other symptoms.

Pitres (*Revue mens.*, 1877, Dec., p. 902) reports a case which *Charcot* diagnosed as one of spastic paralysis, but which afterwards developed other symptoms. On autopsy multiple sclerosis was found. Both anterior pyramids and foci in the lateral columns were sclerotic. *Hydromyelus of the cervical portion* of the cord with *symmetrical* (*secondary?*) *degeneration of the posterior portions of the lateral columns* in the lumbar portion of the cord, were found in a case of spastic paralysis, in which later disturbances of sensation occurred (*Sänger*).

Shaw reports a case giving a typical clinical history of bulbar paralysis with progressive muscular atrophy, *without contracture,* in which, in addition to the ordinary lesion of the medulla and anterior horns, sclerosis "of a very light character throughout the cord," was found in the lateral columns.

Two cases in which the symptoms of spastic paralysis were prominent, though complicated by others, in which *no lesions of the lateral columns existed,* are of special interest. *Schulz* reports a case of *internal hydrocephalus* of twenty-five years' duration, in which some disturbance of sensation was the only symptom complicating an otherwise typical history of spastic paralysis. The cord was normal.

Mader reports a case in which the *white substance of the cord was normal.* The prominent spastic symptoms which existed in life seemed to have been caused by a *lesion of the cauda equina.* The anterior gray columns were sclerosed in part, and a peculiar, apparently inflammatory degeneration of the muscles existed. Inflammatory changes in the bones and knees were also found. Other symptoms besides those characteristic of spastic paralysis existed during life. A case reported by *Bramwell*, in which rigidity and paralysis of the legs, with numbness and greatly increased tendon-reflex, disappeared after the discharge of a lumbar abscess, may be ranked with this case as being possibly of peripheral origin.

The cases of *combined-system diseases of the cord*, above referred to, must be here alluded to, as in them spastic symptoms were associated with lesions of the lateral columns, *except* in *Westphal's* and *Leyden's* cases.

With reference to the *conclusions to be drawn* from clinical and pathological experience in respect to spastic paralysis, *Leyden*, writing in 1879, states that while the symptom-complex, known as *spastic paralysis*, is pretty frequent in spinal diseases of different kinds, it does not form a peculiar disease in itself. It occurs in some forms of meningitis and myelitis, which are susceptible of cure. He does not think that any decided relationship to sclerosis of the lateral columns is manifested in cases of chronic myelitis, in which the peculiar symptom-complex occurs, although in these cases the periphery of the anterior and lateral columns is, to a greater or less extent, affected. The spastic phenomena can be explained by the interruption of the conduction of motor impulses from the brain by the myelitic process, and the consequent increase of reflex irritability below the lesion. Other influences, however, may cause muscular contraction, such as associated movements from irradiation of voluntary impulses and descending neuritis and myelitis. *Erb* explains the absence of spastic paralysis in those complicated cases reported by *Leyden* and *Westphal*, by the coincident existence of degeneration of the gray substance and of the anterior roots, which must, of course, prevent contracture as well as increased tendon reflex. *Westphal* explains their absence, in his cases, by the implication of the white matter in the posterior root-zone, which prevented reflex action. *Erb* states that *complete* degeneration of the pyramid columns must cause only *paralysis*, since some power of conduction is necessary for the production of spastic symptoms. He concludes (1878) that a decided connection is manifested between sclerosis of the lateral columns and spastic paralysis. Cases recorded since 1878 seem to support his conclusion, although the peculiar symptom-complex of spinal paralysis has been exceptionally found *without* lesion of the lateral columns. These cases were not, however, perfectly typical.

Erb and *Charcot* lay great stress on the necessity of limiting the designation *spastic paralysis* to those cases which are *perfectly* typical. Any complicating symptom (disturbance of sensation, vesical weakness, etc.) throws doubt on the diagnosis. The history of the recently reported complicated cases illustrates fully the truth of the latter statement. The possibility that certain diseases of the brain may commence, especially in children, with the symptoms of spastic paralysis is to be kept in mind (see case of *Schulz* and clinical observations of *Seguin* and of *Miles*, spastic infantile paralysis, *Med. Rec.*, N. Y., 1879, XVI., 217).

Stümpell calls attention to the *relaxation of the limbs*, which takes place in spastic paralysis, when the legs are deprived of the irritation of their own weight by the support afforded by water in a bath. The use of the bath is of value in determining the amount of paralysis, the spastic element being thus eliminated. The same observer saw *ankle-clonus* in typhoid fever and in phthisis without other nervous symptoms, except, in some cases, hyperæsthesia of the muscles. No change was found in the cords of the phthisis cases to explain the phenomenon. He found increased tendon reflex in a case of lead paralysis affecting only one arm, in a case of poisoning by strychnia and in three cases of poisoning by atropine.

TREATMENT AND PROGNOSIS.

Mitchell saw a case of amyotrophic lateral sclerosis improve in all respects under tonic treatment (elixir quiniæ, ferri et strychniæ, phosphates and cod-liver oil). He saw two cases resembling spastic paralysis recover in sixteen hours and three weeks respectively. *Henck, G.*, had a case, very nearly typical, which recovered in thirty-four days. Dry cups to the spine and purgatives were used. *Velden* gives one of recovery after thirteen months' illness. Case was not quite typical. Chloride of gold was used for two and a half months previous to recovery (chloride of gold, 0.3; distilled water, 15.00; fifteen to twenty drops three times a day). *Mitchell* obtained temporary relaxation of spastic contraction by the use of *massage*. In the absence of the spastic contraction, the patient could not stand up. No other cases are reported which shed light on the treatment or lighten the prognosis of this class of diseases.

HEMIPLEGIA AND HEMIPARAPLEGIA SPINALIS.

BIBLIOGRAPHY.

WEISS, R: Stich an's R.-M. zwischen Atlas u. Schädel. Durchtrennung der rechten Hälfte. Langenbeck's Arch., XXI., S. 226, 1877.—GOWERS: Unilateral Gun-shot Injury to the Spinal Cord. Lancet, 1877, Nov. 17th, p. 728.—REMAK, E.: Ein Fall von atropischer Spinallähmung durch traumat. halbseit. Blutung in. d. Halsanschwellung des R.-M. Berl. klin. Woch., 1877, Nr. 44.—SCHULTZE, F.: Beitr. zur Lehre von den Rückenmarkstumoren. Arch. f. Psych., VIII., S. 367, 1878.—HENRY, JAMES: A Case of Spinal Hemiplegia. Am. Jour. of Med. Sci., Oct., 1877, p. 440.—VULPIAN, A.: Myélites chroniques, compression lente de la moëlle. Clinique méd. de l'hôp. de la Charité. Paris, 1879, 657, 667.

A short mention of *Gowers'* case is all that calls for special mention here. In this case, a splinter of bone, knocked off by a pistol-bullet, injured the right half of the cord between the second and third vertebræ. Reflex action was diminished on the affected side. The sensibility to pain was abolished, that to touch was retained. The *posterior column was not directly injured.* The anæsthesia reached exactly to the median line.

POLIOMYELITIS ANTERIOR ACUTA.

BIBLIOGRAPHY.

Herrmann, J.: De la paralysie infantile chez l'adulte. Thèse, Paris, 1876.—Salomon, G.: Vier Fälle von spinaler Lähmung Erwachsener. Berl. klin. Woch., 1877, Nr. 39.—Seeligmüller: Ueb. die Unzulänglichkeit uns. Kenntnisse über das Mitralstad. der spin. Kinderlähmung. Amtl. Bericht d. 50. Vers. deutsch. Naturforscher u. Aerzte zu München, 1877, S. 300.—Rosenthal, M.: Zur klin. Charakteristik der Poliomyelitis anterior. Virch. Arch., Bd. 72, 1878.—Schultze: Beiträge zur Pathol. und pathol. Anatomie des centralen Nervensystem, etc. Virchow's Arch., Bd. 73, S. 444.—Althaus, J.: On Acute Anterior Myelitis in the Adult. Am. J. M. Sci., April, 1878.—Seeligmüller: Ueber Lähmungen im Kindesalter. Jahrb. f. Kinderheilk., XII., 321; XIII., 226, 315.—Hamon, Henri: De la paralysie infantile, ses déformations, son traitement. Thèse de Paris.—Althaus, J.: On Infantile Paralysis. London, 1878.—Simon, J.: Diagnostic et traitement de la paralysie infantile. Gaz. méd. de Paris.—Müller, F.: Zur Poliomyelitis anterior acuta bei Erwachsenen. Oester. ärztl. Vereinstg., Wien, 1879, III., 45.—Sturges, W. A.: Three Cases of Acute Anterior Poliomyelitis in Adults. Brit. M. J., 1879, I., 849, 888.—Sainton, H.: Quelques considérations à propos d'un cas de paralysie spinale atrophique aiguë de l'adult. France méd., 1879, XXVI., 514, 523.—Müller, F.: Die acute atrophische Spinallähmung der Erwachsenen. Stuttg., 1879.—Kirmisson, E.: De l'intégrité de faisceau claviculaire du trapize dans la paralysie infantile du membre supérieur. France méd., Par., 1879, XXVI., 673.—Mader: Progressive Myelitis anterior mit Muskelatrophie, Hypoglossuslähmung. Tod.-Ber. d. kk. Krankenanst. Rudolph Stiftung in Wien, '78, 350.

The important recent contribution to this subject is the case of acute poliomyelitis in an adult, with autopsy reported by *Schultze.* The patient was a woman, æt. 42. The disease began with a chill, followed by eight days of fever. The paralysis was typical in its way of occurrence and in the electrical reactions. Exposure to cold was the apparent cause. Death resulted from phthisis pulmonalis in thirty-four days after chill. On autopsy, degeneration was found in the anterior horns of those parts of the cord which corresponded to the affected limbs and degeneration of the corresponding anterior roots. At the junction of the lumbar and dorsal portion of the cord, a number of slightly and a few greatly swollen axis-cylinders among a large number of normal ones were found, predominantly in the antero-lateral columns.

Clinical histories of this disease in the adult, quite typical in their course, are given by *Althaus*, *Sturge*, and *Sainton*.

Seeligmüller calls attention to the small number of exact observations of the *initial stage* of the disease which have been recorded. The initial symptoms are often so slight as not to give rise to careful examination of the cases. His last communication contains an analysis of seventy-five cases in children, which occurred in his own practice. The analysis led to no result which needs to be mentioned here. He records four cases, two of them doubtful, in which *progressive muscular atrophy* occurred in later life in patients who had infantile paralysis.

Kirmisson calls attention to the non-implication of the upper part of the trapezius in two cases of infantile paralysis, in which complete atrophy of the deltoid, biceps, supra- and infra-spinati, and lower two-thirds of the trapezius existed.

Seeligmüller saw complete recovery in one case of infantile paralysis of the right shoulder after four and a half months of electrical treatment.

POLIOMYELITIS ANTERIOR SUBACUTA ET CHRONICA.

BIBLIOGRAPHY.

Déjerine: Atroph. muscul. et paraplég. dans un cas de syphilis maligne précoce. Arch. de Physiol., 1876, 430.—Kétly, K.: Poliomyelitis anterior acuta et chronica. Wien. med. Woch., 1877, Nr. 28 u. 29.—Salomon, G.: Berl. klin. Woch., 1877, Nr. 39.—Eisenlohr: Neuropatholog. Beiträge, I. Zur Casuistik der subacut. vorderen Spinallähmung. Arch. f. Psych., VIII., S. 310, 1878.—Rosenthal, M.: Zur klin. Characterist. d. Poliomyelit. anterior. Virch. Arch., Bd. 72, 1878.—Erb, W.: Ueb. eine noch nicht beschrieb. Mittelform der chron. atroph. Spinallähmung. Vorläufige Mittheil. Erlenmeyer's Centralbl. f. Nervenheilk. u. s. w., 1878, Nr. 3.—Bataille, V.: Contributions á l'étude de la paralysie spinale atrophique de l'adulte. Paris, 1878.—Malone, M. J.: Case of Subacute Spinal Paralysis in the Adult. Dublin J. M. Sci., 1879, 297.—Kahler und Pick: Beiträge zur Lehre von den Veränderungen der elektrischen Erregbarkeit bei Poliomyelitis anterior subacuta. Vrtljschr. f. d. prakt. Heilk., 1879, II., 41-72.—Edes, R. F.: A Case of Anterior Spinal Paralysis with Formation of Vacuoles in the Ganglion Cells of the Spinal Cord. Bost. M. and S. J., 1879, CI., 105-109.—Renaut: Remarques anatomiques et cliniques sur deux points particuliers de l'intoxication saturnine chronique. Gaz. méd. de Paris, 1878, p. 394.—Déjerine: Recherches sur les lésions du système nerveux dans la paralysie diphthéritique. Arch. de Physiol., 1878, p. 107-144.—Aufrecht: Die Ergebnisse eines Falles von subacuter Spinalparalysie, etc. Deutsches Arch. f. klin. Med., Bd. XXII., S. 33.—Kahler u. Pick: Ueber Vacuolenbildung in den Ganglion-Zellen des Rückenmarkes. Vrtljschr. f. d. prakt. Heilk., 1879, Bd. II., S. 5-17.—Vulpian: Paralysie atrophique de l'enfance. Chir. méd. de l'hôp. de la Charité, Par., 1879, 772–779.—Remak, E.: Ueber die Localisation atrophischer Spinallähmung und spinaler Muskelatrophieen. Arch. f. Psych., 1879, IX., 576.—Bernhardt: Beiträge zur Pathol. der peripherischen und spinal. Lähm. Virchow's Arch., 1879, Bd. 78, 267.—Ricklin: Considérations sur deux cas d'atrophie musculaire sans altération des cornes antérieures de la moëlle. Gaz. méd., No. 49, 50, 1878. — Adamkiewicz, A.: Zwei parallele Fälle, Poliomyelitis, Bleilähmung. Charité-Ann., Berl., 1879, IV., 430, 454.

The etiological connection between *lead poisoning* and poliomyelitis anterior has been investigated by *J. J. Mason* (lead-poisoning in frogs, *N. Y. Med. Jour.*, 1877, July, p. 36). His results were negative. *Erb* alludes to them as needing confirmation. *Vulpian* found chronic myelitis with destruction of the ganglion-cells in a dog poisoned by lead. In a case of lead paralysis in the human subject, he found degeneration and atrophy of isolated ganglion-cells, with some increase of nuclei, and with sclerotic spots in the roots of the cervical enlargement. *Friedländer* (*Virch. Arch.*, 75, S. 24, 1879) records an autopsy of a case of lead paralysis in which degenerative changes were found in the muscles, nerves and spinal roots, but the *spinal cord was perfectly normal. Remak* criticises this case unfavorably on account of the imperfect clinical history.

As shedding some light on the *pathology* of the affection may be cited the cases of *Vulpian* and *Aufrecht*. These were complicated cases of irregularly distributed myelitis in adults, in which atrophy of the muscles and lesions of the anterior horns existed. The latter in his case found certain clear nucleated fibres, with here and there a spindle-formed mass of striated muscular substance, which he regarded as muscular fibres in the process of regeneration. Scattered through the nerves he found small nucleated fibres, in some places split into two parts, which he regarded as newly developing nerve-fibres.

Kahler and *Pick* report a case in which an excessive formation of vacuoles in the ganglion-cells of the anterior horns was the only spinal lesion found to explain paresis without evident atrophy, which existed during life in all the limbs. The only change in the electrical reactions was a diminished excitability to both currents in some muscles, principally in the extensors of the arms and hands. A similar lesion was found in a case reported by *Edes*, in which atrophy of forearms and legs began to develop less than four months before death. The abstract of this case given in the *Boston Med. and S. J.* is very imperfect. *Déjerine* publishes a case which, clinically, was not a typical one of poliomyelitis anterior, but in which degenerative atrophy of the muscles existed with degeneration of the motor nerves and anterior roots. *The ganglion-cells of the anterior horns in the lumbar enlargement had, for the most part, disappeared.* There was no indication of an irritative process in the connective tissue or on the blood-vessels. This same observer, from the examination of five cases of *diphtheritic paralysis*, concludes that in the anterior roots of the spinal nerves a lesion is *always* found, which corresponds to that which occurs in the distal end of a cut nerve, and is proportionate in intensity to the duration of the paralysis. The posterior roots are never affected. A light alteration, to which the above is secondary, is always found in the gray substance, affecting the parenchyma and the interstitial substance equally, and not limited to any particular cell groups.

Ricklin speaks of a case (reported by *Debove* in the *Progrès Méd.*, 1879, p. 856) in which atrophy rapidly involved the legs, the forearms slightly, and the hands considerably, coming on with fever and pain in the limbs. These latter ceased after the second day in the arms, in the legs they diminished in violence, coming on in paroxysms three or four times an hour. Paralysis was nowhere complete. Sensibility and the sphincters were normal. Death occurred in three and a half months from intercurrent pneumonia. On autopsy, *the spinal cord was normal.*

Clinical histories of more or less typical cases are recorded by several observers, which confirm on the whole *Erb's* statements in regard to course and prognosis of poliomyelitis anterior chronica. *Renant* records two cases of *extensive lead paralysis*, which was preceded by *high fever*, with no other apparent origin than the process which resulted in the paralysis. *Bernhardt* records a case of *lead paralysis*, in which the electrical reactions were those of *Erb's middle form* of poliomyelitis anterior (see below).

Erb states that the *fibrillary contractions* of the muscles, which are usually present in the earlier stages of poliomyelitis anterior chronica, may be absent. The atrophying muscles are often painful on pressure, and may also be the site of spontaneous pains. In the more lightly affected muscles, reflex action may be only diminished, not, as is usually the case, entirely abolished.

Cases strongly resembling *lead paralysis* are reported by *Erb*, *Rosenthal*, and *Adamkiewicz*. This latter observer saw a formerly very free *secretion of sweat* disappear with the onset of the paralysis.

Erb has recently described what he calls a *middle form* of chronic poliomyelitis anterior, which differs from the ordinary type of the disease in that the *paralysis is never complete*, that the reflexes are not at all or only partly abolished, and that, while the *degeneraction reaction in the muscles themselves is marked, the nerves are not at all or very little affected.*

Erb has seen four cases of this form of disease. Its course is very gradual. The evenly distributed muscular atrophy develops without fibrillary contractions. In other respects the symptoms are those of typical poliomyelitis anterior chronica. Three of *Erb's* cases improved, one recovering completely. The fourth developed slight symptoms of bulbar paralysis, but seemed to be stationary when it was recorded. The constant current applied over the spine was used in the case of cure.

In this place will be mentioned the results obtained by *Remak*, from a recent clinical study of twenty-one cases, including infantile, lead and chronic atrophic paralysis, the latter including four cases of progressive muscular atrophy. He finds that in these diseases there exists a marked tendency toward the *isolated involvement of certain groups of muscles which act functionally together*, although the individual muscles of these groups are innervated by different nerve-trunks, and although other muscles innervated by these same nerve-trunks are not involved. The most important of these groups are the biceps, brachialis anticus, and supinator longus—the muscles on the dorsum of the forearm, exclusive of the supinator longus—the quadriceps extensor cruris and tibialis anticus, the sartorius and the perinei not being involved. These groupings have been previously observed in isolated cases. *Remak* regards this fact as pointing to the probability of the existence of *certain foci of ganglion-cells in the cord corresponding to these groups of muscles*, and therefore to the probability that any disease in which this grouping occurs is of spinal origin, although an affection of the spinal roots or of the plexuses near the cord may give rise to a similar grouping of affected muscles. He argues that this tendency in lead paralysis is an indication of its spinal origin. In a case reported by *Schultze*, in which all the muscles supplied by the sciatic nerves, except the tibiales antici were paralyzed, on autopsy it was found that the lower half of the lumbar enlargement of the cord was degenerated. The case thus demonstrated that the spinal centre for the tibialis anticus is in the upper half of the lumbar enlargement and disassociated with the centres of the other muscles supplied by the same peripheral nerve.

ACUTE ASCENDING PARALYSIS.

BIBLIOGRAPHY.

VULPIAN: Leçons sur les malad. du syst. nerv. Paris, 1877, p. 188.—GRASSET: Malad. du syst. nerv. Paris, 1878, p. 467.—KÉTLY, K.: Vergiftung mit Sublimat. Tod in Folge von aufsteig. acut. Paralyse. Pester med. chir. Presse, 1878, Nr. 8 u. 9.—JAFFE, T.: Ein Fall von Paralysis ascendens acuta. Berl. klin. Wchnschr., 1878, XV., 653.—DÉJERINE, J.: Sur l'existance des lésions des racines antérieures dans la paralysie ascendante aiguë. Gaz. méd. de Paris, 1878, 375.—Recherches sur les lésions du système nerveux dans la paralysie ascendante aiguë (Paris, Thèse).—FOX, A. W.: Case of Acute Ascending Paralysis. Brain, 1879-80, II., 418.

In *Kétly's* case of acute ascending paralysis following poisoning by corrosive sublimate, the anatomical investigation was negative in results.

The case reported by *Jaffe* occurred in a syphilitic subject after excess in venery. The abolition of all the reflexes, and the almost complete abolition of faradic irritability of nerves and muscles with slight galvanic irritability, seems to point rather to acute poliomyelitis anterior. The reactions were tested by *Erb*. No autopsy.

Fox's case was almost typical in its clinical course. No changes were found in the cord except an apparent slight increase of connective tissue in the cervical region of the cord. Sections of the medulla seemed to stain with abnormal readiness. Some increase of connective tissue seemed to exist about the cauda equina.

Déjerine claims to have found in two cases of acute ascending paralysis seen under *Vulpian,* an alteration in certain fibres of the anterior roots (parenchymatous neuritis). The myeline was broken up into fragments. Multiplication of nuclei in the white substance of Schwann, and disappearance of the axis cylinders were noted. The majority of the fibres were unaltered. The same lesions were found in the intra-muscular nerves of the affected members.

TUMORS OF THE SPINAL CORD.

BIBLIOGRAPHY.

SCHULTZE, FR.: Beitr. z. Lehre von den Rückenmarkstumoren. Arch. f. Psych., VIII., S. 367, 1878.—ROTH, W.: Glyome diffuse de la moëlle, syringomyélie, atrophie musculaire. Arch. de physiol. norm. et path., Par., 1878, 612-647.—KOHTS: Gerhardt's Handbuch der Kinderkrankheiten. Bd. V. (Tubercular Tumor of the Cord).—MADER: Einige Fälle seltener Spinalkrankheiten. Wien. med. Presse, 1879, 1058.

In the cases of *glioma* of the cord reported by *Schultze* and *Roth,* the process seemed to take its point of departure from the ependyma of the central canal of the cord.

Schultze's case, a telangiectatic gliosarcoma myxomatodes, extended from the conus terminalis to the pons. For a while it presented the symptoms of a lesion of one-half of the cord. The peculiar combination of irritative and paralytic symptoms of striking inconstancy is brought forward by *Schultze* as a diagnostic point in tumors of the cord. The other cases reported call for no special comment.

SECONDARY DEGENERATIONS OF THE SPINAL CORD.

BIBLIOGRAPHY.

DÉJERINE, J., et MAYER, A.: Recherches sur les altérations de la moëlle épinière et des nerfs du moignon chez des amputés d'ancienne date. Gaz. méd. de Paris, No. 39-40, 1878.—ISSARTIER: Des dégénerations secondaires de la moëlle épinière consécut. aux lésions de la substance corticale du cerveau. Thèse, Paris,

1878.—TAYLOR, F.: A Case of Disease of the Brain with Descending Degenerations of the Spinal Cord. Guy's Hosp. Rep., 1879, 169–184.

Taylor's case is the only one which calls for special mention. It was one of typical descending degeneration, originating in a peculiar gelatinous transformation of the corpus callosum, portions of the optic thalami, etc. The gelatinous substance of the cerebrum dissolved in alcohol, leaving only the blood-vessels, which showed nothing abnormal except a collection of small bright nuclei on their walls. The degeneration of the cord was darker and more sharply defined than usual. In the degenerated portion a structureless, almost homogeneous tissue took the place of the nerve tissue. The disease followed a fall on the back of the head. Paresis and spastic contraction of the limbs were the spinal symptoms of the case.

SPINA BIFIDA.

BIBLIOGRAPHY.

RANKE, H.: Zur Aetiologie d. Spina bifida. Amtl. Bericht der 50. Vers. d. Naturforscher u. Aerzte, München, 1877, S. 297.—Jahrb. f. Kinderheilk., XII., S. 116, '78.

Ranke advances the theory that *spina bifida lumbalis* is caused by a growing together of the membranes of the cord and the external skin in embryonal life before the closing in of the spinal canal, the closure of the canal being thus prevented. His theory is based on three autopsies, in all of which the lower end of the cord was adherent to the sack.

SYRINGOMYELITIS.

BIBLIOGRAPHY.

SCHÜLE: Beitrag z. Kenntniss der central. Höhlenbildung im R.-M. Deutsch. Arch. f. klin. Med., XX., S. 271, 1877.—PICK, A.: Beitrag zur norm. u. pathol. Anatomie der Centralnervensyst. Arch. f. Psych., VIII., S. 283, 1878.—SCHULTZE, F.: Beitr. z. Lehre von der Rückenmarkstumoren. *Ibid.*, VIII., S. 367, 1878.—KAHLER und PICK: Beitrag z. Lehre von der Syring. und Hydromyélie. Vrtljsch. f. prakt. Heilk., 1879, II., 20.

In *Schüle's* case, an enlargement of the central canal from softening of its walls occurred. The supposition that a congenital hydromyelitis may be the point of departure for a syringomyelitis later in life (*Leyden*) is supported by recent observations of *Pick*, who saw two and even three central canals obviously formed by the growth of the ependyma and by the dividing off of diverticulum-like processes. *Schultze* considered that in his case the same thing might have happened.

Kahler and *Pick* saw in an adult cord a triangular dilatation of the lumbar portion, which was otherwise normal, obviously due to a defect of development. In a second case, the same condition existed, except that there was an increase of connective tissue around it, and the epithelium was in part destroyed. They found dilatation of the central canal in a case of amyotrophic lateral sclerosis and in one of progressive muscular atrophy with marked sclerosis of the posterior columns. They suggest that the condition noted in the first two cases may be regarded as an indication of deficient development, and therefore of predisposition to disease.

TONIC CONTRACTIONS OF VOLUNTARY MUSCLES.

BIBLIOGRAPHY.

SEELIGMÜLLER: Ueber spastische spinale Paralysen bei Kindern. Amtl. Ber. der 50. Vers. deutscher Naturforscher und Aerzte, München, 1877, S. 299.—Ueber spastische spinal Paralyse. Jahrb. f. Kinderheilk., XIII., S. 226.

Seeligmüller has observed two more cases of this rare affection. The first was a female public singer, whose mother had suffered from cramps in the calves. From her earliest childhood spontaneous tonic contractions of the kind described in *Seeligmüller's* first case (see *Erb*) would occur at times in the muscles of the calves, face, tongue, and hands. Otherwise she was perfectly healthy. The muscles of the legs, thighs, and upper arms were hypertrophied. Treatment effected very little, but some spontaneous improvement took place after its cessation.

The second case was a strongly developed healthy man, aged twenty-eight years, without hereditary tendency to disease. During four or five years he had stiffness all over the body. If he sat still for a while he would have to stretch and press his knees together before he could rise. If he wanted to dance he would have to try for some time before he could get going, and then there was no further difficulty. If any muscle was caused to contract by electricity it would not relax immediately. The right leg, right gluteal region, and the muscles of the right side of the back was more developed than those of the left side. Treatment with the induced current produced no effect. Massage caused some improvement.

VASO-MOTOR AND TROPHIC NEUROSES.

GANGRENE.—ANGINA PECTORIS.—EXOPHTHALMIC GOITRE.—PROGRESSIVE MUSCULAR ATROPHY.—PSEUDO-MUSCULAR HYPERTROPHY.—EPILEPSY.—TETANUS.—PARALYSIS AGITANS.—ATHETOSIS.—CHOREA.—HYSTERIA.

BY

JAMES J. PUTNAM, M.D.,

OF BOSTON,

PHYSICIAN TO OUT-PATIENTS, MASS. GEN. HOSP.; LECTURER ON DISEASES OF THE NERVOUS SYSTEM, HARVARD UNIVERSITY.

MIGRAINE.

BIBLIOGRAPHY.

Fischer: Exp. Studien zur therapeutischen Galvan. des Halssympathicus. Deutsch. Arch. für klin. Med., XVII., 1875.—Ringer: British Medical Journal, 1874, 725.—Schulz, H.: Allg. med. Centralz., 1875, 61.—Randon: Revue de thér. méd. chir., Feb., 1876.—Chaumier, E.: Un chapitre de l'histoire des maladies constitutionnelles, la migraine. 4to, Paris, I., Thèse No. 358, 1878.—Schumacher: Ergot in the Treatment of Angioparalytic Megrim. Lancet, London, 1878, II., 212-213.—Seuere, I.: Du traitement de la migraine par le chloral. Bull. gén. de thérap., Paris, 1878, XCV., 365-367.—Schwarz, B.: Ein Fall von scheinbarer Hemikranie mit tödtlichem Ausgange. Wien. med. Presse, 1878, XIX., 270-271. —Bonnal, L. A.: Migraine ophthalmique datant de vingt-cinq ans caractérisée par des accès douloureux avec symptomes epileptoides traitée et guérie par le bain d'air chaud. Rev. mens. de méd. et de chir. Paris, 1878, II., 279-282.—Galezowski: Etude sur la migraine ophthalmique. Arch. gén. de méd., Paris, 1878, CXLI., 669; 1878, CXLII., 36-56.—Ketli, K.: Hemikranie bei einem syphilitischen Individuum; Heilung durch Quecksilber. Pester med. chir. Presse, Budapest, 1878, XIV., 33-36.—D'Ancona, N.: La emicrania in rapporto alla sua terapia. Gazz. med. Ital. prov. Venete, Padova, 1877, XX., 49; 57.—Hache: Migraine héréditaire et constitutionnelle datant de plus de trente ans compliquée depuis deux ans de gastralgie rebelle; traitement par l'électricité statique; guérison. Union méd., Paris, 1877, XXIV., 30, 525-526.—Seguin, E. C.: A Contribution to the Therapeutics of Migraine. Med. Rec., N. Y., 1877, XII., 774-776.—Mitchell, S. W.: Brit. Med. and Surg. Jour., 1879, 444. Migraine in a Child 7½ years old.

We have only to report as specially important a few points in the *treatment* of this disease. *E. C. Seguin* has called attention anew to the value of cannabis indica, given in the "continued dose" daily, for months together, with the idea of treating, not the paroxysms, but the disease itself. This mode of using the drug was described in 1872 by *Richard Greene* (*Practitioner*), whose statements Dr. *Seguin* reviews and corroborates. The dose for an adult female is 0.02 to 0.03 of the solid extract three times daily; for an adult male, 0.03 to 0.04. From this dose he has seen no unpleasant effects of consequence. This treatment has met with favor at the hands of a number of other practitioners, among them the writer, though no extended account of its use has come to my knowledge. In two cases I have seen an annoying degree of drowsiness follow the use of even the smaller dose, and a less quantity was found sufficient to diminish considerably the number and severity of the attacks. For some cases it is valueless.

Under the head of *ophthalmic migraine* a great variety of ocular symptoms have been described, some of them such as often precede attacks ot ordinary migraine, such as pain, amblyopia, scotoma, photophobia, and hemiopia occurring periodically. For an extended discussion of this subject the reader is referred to papers by *Galezowski* and by *Bonnal.*

The careful experimental work of *Fischer*, as to the effects of galvanization of the sympathetic upon the cerebral circulation, is worthy of study as bearing on the theory of the action of galvanism in migraine. This treatment is probably far less efficacious than was formerly supposed in directly modifying the cerebral circulation. As oberved in animals, such results are extremely slight and often altogether wanting, and at best are not to be brought about by closures or interruptions of the circuit, but rather by letting the current flow. *Eulenburg* also, while confirming his earlier statements as to the therapeutic value of the galvanic treatment of migraine, points out, himself, the significance of *Fischer's* experiments, and says that *Przcewoski* likewise found the cervical sympathetic inexcitable by interruption or closure of the circuit, but responsive to the polar action of the continuous current as evinced by unilateral changes in the temperature of the face, which fell a little when the kathode was applied, and rose, though but very slightly and temporarily, under the influence of the anode (2d edition *v. Ziemssen's* Cyclop.).

ANGINA PECTORIS.

BIBLIOGRAPHY.

Huppert: Reine Motilitätsneurose des Herzen. Berl. klin. Wochenschrift, 1874, S. 19–22.—Cordes: Angina Pectoris Vasomotoria. Deutsches Arch. f. klin. Med., 1874, XIV., S. 141.—Lusting: Zur Lehre von den vasomotorischen Neurosen. Diss., Breslau, 1875.—France médicale, 1875, Nov.—Johnson, G.: On the Relation between Angina Pectoris and Peripheral Arterial Contraction, and on the Modus Operandi of Nitrite of Amyl as a Remedy for the Disease. Brit. M. J., London, 1877, I., 770.—Gairdner: Angina Pectoris and Allied States, including Certain Kinds of Sudden Death. Syst. Med. (Reynold's), London, 1877, IV., 535–599.—Richter, F.: Angina Pectoris als central bedingte Neurose. Deutsch. Arch. f. klin. Med., Leipz., 1877, XIX., 357–365.—Traube, L.: Zur Lehre von der Angina Pectoris. Ges. Beitr. z. Path. u. Physiol., Berlin, 1878, III., 172, 183.—Clark, A.: Angina Pectoris Associated with Disease of the Aorta. Brit. M. J., Lond., 1878, II., 314.—Leroux, C.: Angine de Poitrine, Mort subite, Athérome arteriel, Compression du Pneumogastrique droit. Progrès méd., Par., 1878, VI., 523.—Huchard, H.: Angine de Poitrine et Pulmonaire, Paral. Consée. du Nerf pneumogas. Union méd., Par., 1879, XXVIII., 433–489.—Murrell, W.: Nitroglycerin as a Remedy for Angina Pectoris. London Lancet, 1879, I., 80, 113, 151, 225.

SYMPTOMATOLOGY AND PATHOLOGY.

For an interesting systematic discussion of this disease, among publications later than the first edition of this Cyclopædia, the reader is referred to a paper by *Gairdner* in the fourth vol. of *Reynold's System of Medicine*, which hardly admits of brief analysis. The frequency with which the pain is accompanied or replaced by a sense of indefinable distress, referred, so far as it can be localized, to the chest, but infecting also the patient's mind with a feeling of profound anxiety or a fear of impending death, is strongly dwelt upon.

Gairdner agrees with *Eulenburg* in believing that in fatal cases of angina pectoris there probably is always some organic disease interfering with the structure, nutrition, or innervation of the heart underlying the paroxysms of pain, vaso-motor phenomena, and the other nervous symptoms. The neurosal symptoms may, however, undoubtedly exist by themselves, as is pointed out by *Richter*, and indeed the relation between the organic changes discovered after death and the neuralgic attacks is still unknown, nor is the cause of death always clear. Thus *Leroux* reports a case of sudden death where there was extensive atheroma of the aorta with periarteritis; also hypertrophy of the left ventricle, but no other signs of cardiac disease. It was impossible to trace out the nerves of the cardiac plexus from the midst of the thickened tissue by which they were surrounded, but the fibres of the sympathetic system just above this point were normal. The right pneumogastric was closely adherent to an enlarged gland near a bronchus, but on microscopic examination its structure appeared normal. The coronary arteries were in all essential respects normal.

Richter lays stress on the fact that heart-disease is often entirely absent, and collects the arguments in favor of the view that angina is a primary central neurosis, probably of cerebral origin. Thus he points out that the paroxysms are liable to be brought on or intensified by cerebral excitants, such as tobacco, anxiety, over-work, and that the characteristic symptoms find their analogues in some of the phenomena of hysteria, lesions of the pons, and other central affections.

TREATMENT.

Dr. *George Johnson* discusses the *modus operandi* of the amyl treatment, recommended by *Brunton* and others, and while fully confirming the previous statements as to the value of the remedy, he combats the view that it acts as at first suggested, that is, by diminishing arterial spasm, believing, on the contrary, that it is equally efficacious in cases where the peripheral arteries, instead of being contracted, are dilated, as shown by flushing of the face. He thinks the drug works in virtue of its direct antineuralgic properties, having found it remarkably efficacious in a case of facial neuralgia, and he quotes Dr. *Talfourd Jones* as having had a number of similar experiences. In view, however, of the remarkable fact that nitroglycerin, which closely resembles amyl nitrite in its physiological action on the arterial system, likewise relieves the paroxysms of both angina and trigeminal neuralgias, it seems clear that it is after all the fall of arterial tension, perhaps by modifying the cerebral circulation, which is the active factor in both cases.

Our knowledge of this effect of nitroglycerin in angina is due to *William Murrell*, who took up the experiments of *Field* and others, and also recognized the therapeutic indication to be drawn from them. The action of this drug upon the circulation comes on more slowly than that of amyl (six to eight minutes), and passes off more slowly. It is capable of checking the attack when present, and what is more remarkable, had, in the three cases in which it was used by *Murrell*, a more or less permanent effect. The dose is one to fifteen drops of a one-per-cent solution in alcohol, to be taken three or four times daily for weeks or months.

In experimenting with thirty-five persons, he found a difference in susceptibility, but obtained similar effects in all cases. The unpleasant symptoms are intense throbbing of all the arteries of the body, headache, prostration, even syncope of five or ten minutes' duration. In one case, each dose caused an immediate and rapid secretion of urine. Extended investigations will be looked for with great interest.

EXOPHTHALMIC GOITRE.

(*Basedow's Disease.*)

BIBLIOGRAPHY.

Féréol: Union méd., 1874, No. 153.—Bartholow: Chicago Journal of Nervous and Mental Disease, July, 1875.—Raynaud: Arch. gén. de méd., Juin, 1875, p. 679.—Bulkley: Chicago Jour., etc., Oct., 1875.—Benedikt: Nervenpathologie und Elektrotherapie. Leipz., 1876, II., 1.—Blake, E. T.: Amyl Nitrite in Exophthalmic Goitre. Practitioner, Lond., 1877, II., 189-193.—Yeo, I. B.: A Case of Exophthalmic Goitre with New Phenomena. Brit. M. J., London, 1877, I., 320-322.—Benicke, F.: Complication der Schwangerschaft und Geburt mit Morbus Basedowii. Ztschr. f. Geb. u. Gynäk., Stuttg., 1877, I., 40-42.—Löwenstamm: Struma exophthalmica (Morbus Basedowii). Med. chir. Centralbl., Wien, 1877, XII., 101.—Lacoste, J. B.: Contribution a l'étude du Goitre Exophthalmique. 4to, Par., Thèse, No. 505, 1877.—Sée, G.: Symptômes de la Maladie de Basedow. France méd., Par., 1878, XXV., 689, 697.—Chvostek, F.: Weitere Beiträge zur Pathologie und Therapie der Basedow'schen Krankh. Allg. Wien. med. Ztschr., 1878, XXIII., 33, 87, 238.—Smith, R. S.: Exophthalmic Goitre; Lesions of the Cervical Ganglia. Med. Times and Gaz., Lon., 1878, I., 647-9.—O'Neill, W.: Exophthalmic Goitre and Diabetes Occurring in the Same Person. Lancet, Lond., 1878, I., 307-309.—D'Ancona, N.: Un secondo caso di gozzo exoftalmusnico guarito colla galvanizzazione del simpatico al collo. Indipendente, Torine, 1878, XXIX., 175, 185.—Cuffer: Début de Maladie de Basedow, sans Goitre, in Exophthalmus; Troubles Vaso-moteurs Prédominants du Côté Gauche Palpitations. France méd., Par., 1878, XXV., 442-443.—Duroziez, P.: Du Souffle des Artères cardiaques dans le goitre exophthalmique. Gaz. méd. de Par., 1878, VII., 4, S. 540-542.—Becker, O.: Der spontane Netzhautartierenpuls bei Morbus Basedowii. Klin. Monatsbl. f. Augenheilk., 1880, XVIII., S. 1.

SYMPTOMATOLOGY.

The observations of the past few years have enriched somewhat our data from which to study this mysterious disease, without, however, bringing us materially closer to a satisfactory explanation of its phenomena. Thus it appears (*Yeo, Cuffer*) that the exophthalmus may precede the other symptoms by a considerable interval, and may remain for a long time unilateral; also that exophthalmus of one side may occur simultaneously with enlargement of thyroid on the opposite side, a fact also noticed by *E. T. Blake*. In *Yeo's* case, the right lobe of the thyroid first became enlarged, and the left eye prominent; later the left lobe of the thyroid and the right eye became simultaneously involved. In proportion as the eye began to protrude, first on one side, then on the other, the eyelashes and eyebrows fell gradually out. *Yeo* points out the frequency with which obstinate diarrhœa is observed in these cases as a significant symptom, and *O'Neill* records an observation in which the cardinal symptoms were associated with diabetes of a marked type, both diseases making their appearance together, and leading to the patient's death.

In a case reported by *Fuller*, one symptom is noticed which has been observed by other writers, and also by myself, but is not mentioned by *Eulenburg*. This is a change in the pitch of the voice, which becomes like that of a child, possibly on account of pressure upon the recurrent laryngeal by the enlarged thyroid, but more probably from derangement of the central innervation.

The second edition of the Cyclopædia contains the following (*Eulenburg*): "In a case reported by *Féréol*, besides the cardinal symptoms (struma of the right side, exophthalmus, palpitation), headache, nausea, vertigo, and tremor were present, as well as unsteadiness of the gait, with tendency to fall to the right, and diplopia, traceable to paresis of the right trochlearis. There was, moreover, diminution of the muscular power on the right side of the body, with hyperalgesia, while on the left side there was hypalgesia, and on both sides increased reflexes. I have myself seen a case in which there was paralysis of the abducens, and another in which paresis of the lower extremities was present."

"*Raymond* has seen in four cases the discoloration of the skin known as leucoderma or vitiligo, and quotes *Trousseau* as reporting another of the same kind. The latter writer, as well as *Stellwag* (cited by *Neumann*), *Bartholow*, and *Bulkley*, have observed the occurrence of urticaria or a very similar affection in the course of the disease."

The spontaneous pulsation of the retinal arteries is sometimes present, sometimes missed, and if, as *Becker* believes, it is of nervous origin, it may perhaps come and go like the other symptoms. Of seven cases observed recently by *Becker*, it was present in all but one, at the time of his examination.

Our knowledge of the *pathogenesis of the disease* has been materially advanced by an experimental research of *Filehne's*. This has shown that, in rabbits, the section of the anterior portion of the restiform bodies, without further injury of the medulla, is capable of exciting two of the classical symptoms of the disease, namely, the rapid action of the heart and the exophthalmus, and, though rarely, even the enlargement of the thyroid in addition. That this rapid pulse was due to destruction of the vagus-tract was shown by the fact that, after the operation, neither excitation of centripetal nerves would slow the pulse in the usual reflex manner, nor section of both vagi quicken it further. That the exophthalmus, on the other hand, was not due simply to injury of the sympathetic system of fibres is also evident, since it could be induced even when the cervical sympathetic had been previously destroyed. It was less easy to excite this symptom than that of the rapid pulse. The enlargement of the thyroid was brought out only once and then by an extensive galvano-caustic operation upon the corpora restiformia. The immediate cause of the exophthalmus was believed to be vascular engorgement.

These observations would remove the primary lesion of the exophthalmic goitre from the cervical sympathetic, and refer it to the medulla oblongata, where it probably belongs. Even were there no experimental evidence for this view, it would be easier of acceptance than the others, since it is more readily conceivable that a disturbance of the complex co-ordinating centres and tracts of the medulla oblongata should give rise to this curious array of symptoms, than that they should follow lesions of the sympathetic nerves themselves, whose functions are simpler, and not of such a character that any single influence of either a paralyzing or irritating nature would readily explain the phenomena actually met with.

PATHOLOGICAL ANATOMY.

With regard to the lesions of the nervous system, *R. S. Smith* reports a case in which the lower left cervical ganglion was found to have been destroyed and replaced by a calcareous mass. The nerve-cells in the other ganglia looked somewhat shrunken, but it was thought, with reason, that this might be attributed to the action of the chromic acid in which the specimen had been hardened. *Wilheim* found in another case degenerative changes in one of the upper cervical ganglia.

TREATMENT.

Further testimony as to the value of mild applications of electricity in the cervical region is given by *Chvostek* and by *Wilheim*; while *Sée* vaunts the combined use of hydropathic measures and veratrum viride (gtt. x.–xx. of the tincture daily in divided doses, continued for weeks and months. Compare Cyclop.).

E. T. Blake records improvement in the subjective and cardiac symptoms in one case from the continued use of amyl nitrite (gtt. $\frac{1}{10}$ – ii., 3 t. d. on sugar), but corroboration of the value of this treatment is wanting.

Ringer quotes *R. T. Smith* as having had excellent results in two cases from the continued use of tinct. belladonnæ (♏ v.). The local treatment of the goitre by injection, galvano-puncture, etc., is not to be recommended.

PROGRESSIVE MUSCULAR ATROPHY.

BIBLIOGRAPHY.

Reschansky: Beitr. zur Lehre von der progr. Muskelatrophie, Königsb., 1874. —Poncet: Gaz. méd., 1875, No. 25.—Balmer: Archiv d. Heilk., XVI., S., 327.—Vaeter, von Artens: Allg. Wiener med. Z., 1875.—Erb: Arch. f. Psych. und Nervenkrankh. V., Heft 2.—Lubimoff: Arch. de phys. normale et pathol., 2, sér. I., 1874, p. 884.—Pierret and Troissier: Ibid., 1875, No. 5, p. 735.—Charcot and Gombault: Ibid., 1875, No. 5, p. 739.—Berger: Ein Fall von Sclerosis lateralis amyotrophica. Deutsche Zeitschr. f. pract. Med., 1876.—Lüderitz: Beitrag zur progressiven Muskelatrophie. Diss., Jena, 1876.—Renaut and Debove: Gaz. des hôp., 1876, No. 22.—Pierret, A.: Note sur un cas d'atrophie musculaire progressive caractérisée au début par de la rétropulsion irresistible. Rev. méns. de méd. et de chir., Par., 1877, I., 413–424.—Pick, A.: Zur pathologischen Anatomie der progressiven Muskelatrophie. Prag. med. Wchnschr., 1877, II., 758–763.—Nunn, W. T.: Sections of Spinal Cord and of the Phrenic Nerves in the two Forms of Progressive Muscular Atrophy. Tr. Path. Soc., Lond., 1877, XXVIII., 9.—Dieulafoy: De l'atrophie musculaire progressive et de la sclérose laterale amyotrophique. Gaz. hebd. de méd., Par., 1877, 2 s., XIV., 789–805.—Worms, J.: Note sur un cas d'atrophie musculaire progressive avec paralysie glosso-labio-laryngée. Arch. de physiol. norm. et path., Par., 1877, 2 s., IV., 706-717.—Weiss, N.: Ueber progressive Muskelatrophie, Wien. med. Presse, 1877, XVIII., 691. —Lichtheim: Progressive Muskelatrophie ohne Erkrankung der Vorderhörner des Rückenmarks. Arch. f. Psychiat., Berl., 1878, VIII., 521–548.—During, L.: Ueber die progressive Muskelatrophie und ihr Verhältniss zur progressiven Bulbärparalyse. 8vo, Erlangen, 1878.—Vergenes, J.: De l'adispose sous-cutanée dans ses rapports avec les atrophies musculaires (Valeur sémeiologique de ce signe), 4to, Paris, 1878, No. 314.—Cullingworth, C. J.: Clinical Study of a Case of Progressive Muscular Atrophy, Affecting chiefly the Trapezius, Rhomboids, and Latissimus Dorsi. Med. Times and Gaz., Lond., 1878, II., 121–125.—Schultze: Ueber die Beziehungen der progressiven Muskelatrophie zur Pseudohypertrophie der Muskeln. Arch. f. Psychiat., Berl., 1878, IX., 169.—Steffanini: Caso d'atrofia musculare progr. con alterazione di ganglia simpatici. Arch. delle science med., II., No. 3, 1878 (J. bericht v. Virchow u. Hirsch, 1878, II., 114).—Shaw, J. C.: Progressive Muscular Atrophy and its Pathology. Proc. M. Soc. County Kings, Brooklyn, 1878, III., 46–49.—Rumpf, H.: Zur Function der grauen Vordersäulen des Rückenmarks. Arch. f. Psych., etc., X., 1, 115.—Coredoin, J.: Etude clinique de la paralysie spinale aiguë et de l'atrophie musculaire progressive chez le même individu. 4to, Paris, 1879, No. 584.—Leyden: Ueb. Bulbärpar., etc. Arch. f. Psychiatrie, VIII., 641.—Charcot (Brissaud): Sclérose latérale amyotrophique. Prog. méd., 1880, Nos. 1 and 3.—Debove: (Rapid musc. atr., without lesions in the cord or nerves.) Prog. méd., 1878, No. 45.—Eisenlohr, C.: Idiopathische Muskellähmung u. Atrophie. Cbl. für Nervenh. kunde, 1879, No. 5.

PATHOLOGICAL ANATOMY AND PATHOGENESIS.

The old discussion as to whether this is a disease of myopathic or of neuropathic origin, which for a time had lulled, has recently sprung up afresh apropos of a case reported by *Lichtheim*, where wide-spread muscular atrophy of progressive character, beginning in the muscles about the shoulder, and apparently excited by a local strain, was found after death to be unassociated with any pathological change worth mention, either in the peripheral nerves or the spinal cord. Regarding this case as a typical one, *Lichtheim* concludes that the changes in the ganglion-cells in the anterior cornua of the spinal cord do not necessarily constitute the essential primary lesion of progressive muscular atrophy. The justice of this conclusion is not, however, universally admitted. Among others, *Erb* and *Schultze* deny our obligation to accept *Lichtheim's* as a typical case, saying that neither the clinical history nor the microscopic appearance of the muscles was absolutely pathognomonic. Thus the traumatic origin of the attack suggests that the atrophy may possibly have been secondary to articular disease in the shoulder at the outset, though it appeared later to run a typical course, and the case was an unusual one in several minor respects, such as in the occurrence of paresis of some of the facial muscles, without, however, the usual symptoms of bulbar paralysis, with which, as is well known, progressive muscular atrophy is so apt to be complicated. Still, even the authors cited admit the importance of *Lichtheim's* observation, as well as the fact that we must improve our means of diagnosis before we can distinguish clinically such cases as these from the typical forms of the disease. This, they think, may eventually be possible through careful electrical tests. With regard to the muscular changes, they claim that in true progressive muscular atrophy, the atrophy is characterized by acute degenerative processes, such as multiplication of nuclei, disappearance of the transverse striation, waxy degeneration, etc., whereas *Lichtheim* observed only a simple atrophy of the muscular fibres, with increase and fatty degeneration of the interstitial connective tissue. The importance of this criticism may, in its turn, be called in question.

Thus *Leyden* (Klinik der R. marks-Krankheiten) thinks we are still far from being able to dogmatize about the nature of the typical histological changes. That the symptoms of progressive muscular atrophy are almost invariably associated with, and often distinctly due to degenerative lesions of the great nerve-cells in the anterior cornua of the spinal cord no one to-day can doubt, so that, as *Erb* remarks, it has become almost superfluous to publish further illustrative cases of this kind. On the other hand, it is evident that the group of cases hitherto classed under this name, while they join hands with the recognized amyotrophic spinal affections on the one hand, do so none the less with the primary myopathic diseases on the other, to which the case of *Lichtheim* and those known as pseudohypertrophic paralysis may belong, and it is probable that this element has not of late years received due recognition. It will be very satisfactory if it turns out, as *Rumpf* suggests, that the presence of the degenerative reaction of the muscles to galvanism may serve to mark out a doubtful case as being of neuropathic origin. *Erb* has found this reaction in a few cases of progressive muscular atrophy, and is inclined to think that it is always to be discovered in certain muscles and at certain stages of the disease, if searched for with sufficient care. The difficulty of recognizing it is sometimes increased by the fact that the feeble and slow contraction of the diseased fibres may be masked by the more rapid and vigorous contraction of relatively healthy parts of the same muscle.

Other good observers have searched for this degenerative reaction in vain, so that its value as a sign of neuropathic, as distinguished from myopathic, muscular atrophy is not definitely settled. The discussion as to the pathological anatomy of progressive muscular atrophy would be simplified if all observers would, with *Leyden*, agree provisionally to consider in a group by themselves the cases of so-called "hereditary" muscular atrophy. The disease in these cases, affecting, as it does, the lumbar muscles and those of the lower extremities before those of the arms and hands; making its appearance usually in childhood; and often in several members of the same family, either in the same or in successive generations, appears more closely related to pseudo-muscular hypertrophy (v. next chapter) than to the typical progressive muscular atrophy of adults. Furthermore, the examination of the spinal cord, in "hereditary" muscular atrophy as in pseudo-hypertrophy, has as yet given negative results, though the number of autopsies (one by *Meryon* and several by *Friedreich*) are as yet too few to be conclusive. It is to be remembered that the term "hereditary" for some of these cases is, if literally taken, a misnomer, since it is by no means always possible to find evidence of the existence of the same disease in former generations. All that the word should be taken to imply is, that the causes of the disease in a given case are not post-natal, but pre-natal in the widest sense of the term.

Möbius believes, in spite of the negative results of post-mortem examination, that the cause of the muscular atrophy, even in the "hereditary" cases, is to be sought in the central nervous system, mainly because in these cases there are so often other signs of disturbance in the functions of the brain and cord, etc. To form a decided opinion on this point would be premature.

It is difficult to say through just what agency the lesions in the anterior cornua of the cord act upon the muscles in progressive muscular atrophy, so as to excite inflammatory and degenerative changes in them, even if we grant, with the vast majority of observers, that this actually happens in the great bulk of the cases. It is evident that the lesions beginning in the ganglion-cells are not always propagated per continuum through the peripheral nerves, for in some cases—that of *Erb* and *Schultze* for instance—the motor nerves have been found intact.

The hypothesis of special trophic cells has not met with general favor, but recently *Rumpf*, in an able paper, has defined more physiologically and clearly than has been done before, the view that the motor nerve-cells may lose so much of the trophic influence of which they are supposed to be the seat as to permit the muscle to degenerate, and yet retain enough to nourish passably the nerves by which the two organs are united.

More prudent and philosophical than either of the extreme views (myopathic and neuropathic) as to the primary lesion of progressive muscular atrophy seems to me the opinion or hypothesis expressed by *Leyden* (Klinik der R.marks-Krankheiten, Bd. II., p. 506), as follows: "I regard this disease, therefore, as a process of progressive degeneration . . . which attacks the muscular system, the motor nerves, the motor ganglion-cells, and the motor nerve-tracts in the spinal cord, parts which are all functionally associated, and finally, in its fullest development, involves and destroys the motor nuclei of the medulla oblongata" (bulbar paralysis).

The pathological relations of the amyotrophic diseases of neuritic origin, important as the subject is, cannot be discussed here. They are treated of at length by *Leyden* in a recent exhaustive paper (*Zeitschrift für klin. Med.*, I., 3, 1880).

With regard to the implication of the *sympathetic system* in progressive muscular atrophy nothing is to be added which should change the conclusions arrived at by *Eulenburg* in the first edition of this Cyclopædia. One case by *Steffanini* (known to me only through a reference in *Virchow* and *Hirsch's* J.bericht, by *Bernhardt*) is of interest because: besides pigmentary and fatty changes in the sympathetic ganglia, the spinal cord is said to have been found in a healthy state.

PSEUDO-MUSCULAR HYPERTROPHY.

BIBLIOGRAPHY.

Korwatzki: Ueb. die Hypertr. d. quergestreiften Muskelgewebes, Erl., 1874.—**Ord, W. M.**: Case of Ps.-m. H., with a few Observations on Surface Thermom. Med. Chir. Tr., LX.—**Clarke and Gowers**: Med. Chir. Trans., 1874, p. 247. Bruenniche Hosp. Tid. 2 R., 1 Aarg., p. 257.—**Mahot**: De la par. pseudo-hypertr. Thèse de Paris, 1878 (3 cases; lack of mental power; also commented on by Bäg).—**Björnstrom, F.**: Upsala läkara fören. förhandl., Bd. XII.—**Bäg**: Hosp. Tid., 2 R., Bd. 4.—**Brieger**: Deutsch. Arch. f. kl. Med., 1878.—**Möbius, Samuel**: Kl. Vorträge, No. 17. (Relation between P.-m. H. and Progr. M. Atr.)—**Kesteven**: Jr. of Ment. Sc., Oct., 1879.—**Bennett, A. Hughes**: P. M. H. in an Adult. Brain, Vol. 2, No. 3.—**Goetz, R.**: Beitr. zur Atr. M. Lip. Aerztl. Intell. Bl., 1879, No. 39. (10 cases; etiology: dampness and insufficient nourishment.) **Webber, S. G.**: Pseudo-hyp. Par. Boston M. and S. Jr., 1879, 460.—**Gowers, W. R.**: Pseudo-h. Par. London, 1880.

PATHOLOGY.

The latest facts with regard to this disease are well summed up in a recent monograph by *Gowers*. Of the 220 cases there analyzed, 102 were apparently isolated, and 118 were grouped in 39 families.

Six new autopsies have been made since the publication of the first edition of the Cyclopædia, viz., by *Clarke* and *Gowers*, *Brieger*, *Bäg*, *Schultze*, *Kesteven*, and *Goetz*.

In *Clarke* and *Gowers'* case, there was found extensive "granular degeneration" of the interstitial tissue here and there throughout the entire spinal cord, but especially in the lower dorsal region, where in each half of the gray substance there was an area of disintegration amounting to an actual cavity outside of each posterior vesicular column.

At the lower end of the conus medullaris one group of nerve-cells was wanting. The great nerve-cells of the anterior cornua were, however, "conspicuously healthy," the lateral columns essentially normal, and the changes found are ascribed by the authors to either ascending neuritis or to repeated venous congestion, for which opinion the grouping of the lesions around the posterior nerve-roots and the blood-vessels, as well as the history of the patient, afforded good grounds.

In none of the cases were changes in the ganglion-cells analogous to those found in progressive muscular atrophy described, though it is worthy of note that *Schultze* and *Barth* speak of the number of these cells as being strikingly small—a condition which may have been due to the same remote cause with the impairment of the muscular development, without any closer association existing between them. In *Gowers'* case, however, the number of the cells is expressly stated to have been normal. *Bäg* found signs of degeneration in the lateral columns of the cord. In *Kesteven's* case, besides wide-spread vascular changes, there were small, whitish spots scattered through the brain and cord, within the limits of which the nerve-fibres seemed to have been almost wholly destroyed; and the ganglion-cells, though as a rule normal, were here and there somewhat granular and pigmented. *Goetz* (description known to me only through an abstract in the *Cbl. für med. Wiss.*, by *Bernhardt*) found the ganglion-cells healthy, but speaks of a gelatinous connective tissue present both in the white and gray substance, by which the nerve-fibres seemed to have been compressed.

Eulenburg joins with *Friedreich* in describing the disease pathologically as a *chronic myositis with hyperplasia of the interstitial connective tissue*, a designation which both consider to cover also the muscular changes in progressive muscular atrophy.

Gowers appears to incline to the view that the substance by which the muscle is replaced is a sort of pathological new-growth, a myo-lipoma, indicating "a congenital nutritive and formative weakness of the striated muscular substance," rather than as simply the result of inflammation. In this connection he describes a curious tumor found by him attached to the conus medullaris of the spinal cord (not in a case of this disease). It appeared at first to be made up of fat-cells alone, but on examination proved to contain muscular fibres and connective tissue as well, closely resembling a fragment of pseudo-hypertrophic muscle. Yet it had formed entirely apart from the surroundings and influences which have been supposed to give rise to the disease in question.

Great interest attaches to the discussion as to the relation which pseudo-muscular hypertrophy bears to progressive muscular atrophy.

As was observed in the chapter on the latter disease, this discussion would be simplified if the clinical distinction between the usual adult form of the affection and the so-called "hereditary" form were, at least provisionally, recognized by all.

The likeness of some cases of the disease before us to cases of this last-named division of progressive muscular atrophy is strong reason for considering them as belonging in one group, especially when we reflect that enlargement of muscles is not pathognomonic of pseudo-muscular hypertrophy, since it is occasionally met with, and that, too, pre-eminently in the calves of the legs, in cases of progressive muscular atrophy, both as a result of hyperplasia of the fatty and connective tissues, and in consequence of a true muscular hypertrophy (*Leyden*, "hereditary" form); and further, that the enlargement in pseudo-muscular hypertrophy may give place to a diminution in bulk, and vice-versâ, in the course of the disease.

As for the relation between our disease and the typical adult form of progressive muscular atrophy, this is certainly very remote, even if indeed it exists at all.

For reference to other cases of interest see the bibliographical index.

EPILEPSY.

BIBLIOGRAPHY.

Lasègue, C.: De l'épilepsie par malformation du crâne. Ann. méd. psych Paris, 1877, XVIII., 5 s., 161-178. Also Arch. gén. de méd., Paris, 1877, II., 5-20.—**Vielle, A.**: Quelques considérations sur l'épilepsie et plus spécialement sur l'épilepsie associée avec certaines malformations du corps en général et de la tête en particulier. 4to, Paris (Thése No. 237), 1878.—**Garel, J.**: Recherches cliniques et statistiques sur la valeur de l'asymétrie faciale dans le diagnostic de l'épilepsie. Lyon méd., 1878, XXVII., 5-43.—**Magnan**: Des rapports entre les convulsions et les troubles circulatoires et cardiaques dans l'attaque d'épilepsie. Gaz. méd. de Paris, 1877, VI., 390.—**Westphal**: Eigenthümliche mit Einschlafen verbundene Anfälle. Arch. für Psychiatrie, etc., B. 7, p. 632.—**Fischer, F., Jr.**: Epileptoide Schlafzustände. Arch. f. Psychiatr. u. Nervenkrankh., Berlin, 1877, VIII., 200-203.—**Bouchut**: De l'épilepsie chez les enfants et des vertiges épileptiques. Gaz. d. hôp., Paris, 1877, L., 66-73.—**Bourneville**: Compte rendu des observations recueillies à la Salpétrière, concernant l'épilepsie. Progrés méd., Paris, 1877, V., 623, 643, 655.—**Huppert, Max**: Die Albuminurie in dem epileptischen und paralytischen Anfall. Arch. für Psychiatrie, etc., 1877, p. 189.—

MARTIN, H.: De la mortalité des enfants des épileptiques, etc. Ann. méd. psych., Paris, 1878, XX., 364-368.—DELASIAUVE: Le mariage des épileptiques. Jr. d'hyg., Paris, 1879, IV., 339. JACKSON, J. H.: Lectures on the Diagnosis of Epilepsy. Med. T. and Gaz., London, 1879, I., 223.—SOMMER, WILHELM: Erkrankung des Ammonshorn als ætiologisches Moment bei Epilepsie. Arch. für Psych., etc., X., 631. (Contains full list of references.)—LÉPINE, R.: De l'épilepsie survenant à la suite d'écarts habituels de régime, chez des individus très-sanguins et de son traitement. Rev. mens. de méd. et de chir., Paris, 1877, I., 573-587.—GOWERS, W. R.: On the Use of Iron in Epilepsy. Practitioner, London, 1877, XIX., 241-246.—SCHULTZ: Ein Fall von Hemmung epileptischer Anfälle mit nachfolgender Heilung. Berl. klin. Wchnschr., 1877, XIV., 659-661.—VALLENDER, E.: Coupirung epileptischer Anfälle durch subcutane Apomorphininjectionen. Berl. klin. Wchnschr., 1877, XIV., 185-6.—MARTIN, A.: Dangers des préparations opiacées chez les femmes hystériques. Mouvement méd., Paris, 1877, XV., 488-498.—BOURNEVILLE: Influence de la compression ovarienne sur la contracture hystérique récente. Progrès méd., Paris, 1877, V., 487-488.—PISON, E.: De l'efficacité du bromure de potassium dans le traitement de l'épilepsie. 4to, Paris (Thèse No. 444), 1877.—SEGUIN, E. C.: Report on the Use of Chloride of Potassium in Epilepsy. Proc. Therap. Soc., N. Y., 1878, 21-28. Also N. Y. Med. Jour., April, 1878.—SINKLER, W.: Cannabis Indica in the Treatment of Epilepsy (Case). Phila. Med. Times, 1878, VIII., 607-609.—ROCKWELL, A. D.: On the Use of Electricity in the Treatment of Epilepsy. Med. Rec., N. Y., 1878, XIII., 264-266. Preliminary Report upon the Use of a Mixture of Chloral and Bromides in Epilepsy. N. Y. M. J., 1878, XXVII., 535-539.—AGNEW, D. H.: Successful Case of Trephining for Epilepsy. Phila. Med. Times, 1878, VIII., 578-580.—ECHEVERRIA, M. G.: De la trépanation dans l'épilepsie par traumatismes du crâne. Arch. gén. de méd., Paris, 1878, CXLII., 529, 652.—POIRIER, P.: Des moyens d'arrêter les attaques hystéro-epiléptiques, et en particulier, de la compression des ovaires. Progrès med., Paris, 1878, VI., 993-994.—SPITZKA, E. C.: Some Remarks on the Treatment of Epilepsy and Epileptifrom Conditions. Physician and Pharmac., N. Y., 1879, n. s., XII., 247-249. —KUNZE, C. F.: Ueber die Behandlung der Epilepsie mit Curare (Ber. d. 51. Verhandl. deutscher Naturf. u. Aerzte in Cassel).

ETIOLOGY.

In a valuable series of Gulstonian lectures, Dr. *W. R. Gowers* has given some important statistical information based on the careful analysis of 1,450 cases observed by himself, from which the following facts are taken with regard to *hereditary transmission of the disease*, which, in the main, bear out the views expressed by *Nothnagel*.

In the inquiry, certain affections believed to be related to epilepsy, viz.: insanity, hysteria, chorea, paraplegia, and infantile paralysis occurring in near relatives, are admitted as indications of transmissible taint. In this sense, thirty-six per cent of the cases showed evidence of neurotic inheritance, and this was true of the cases of hystero-epilepsy in about the same proportion with those of true epilepsy.

The females of the family suffer from this influence more than the males (fifty-seven against forty-three per cent), but, nevertheless, when the disease is inherited from the father's side (which is relatively uncommon), the reverse is true, the liability of males being twenty-two per cent greater than when it comes from the mother's, while the mother's taint is transmitted much oftener to girls.

"We may now see, in part at least, why females preponderate so much more in cases of inherited disease than when there is no inheritance. First, the inheritance is more frequent from the mother's side than from the father's, and secondly, when the inheritance is from the mother's side, girls suffer in a much larger proportion than boys."

As to the nature of the disease in the parents from whom epilepsy seemed to have been inherited, the same affection was present in the large majority of instances, insanity in some form being the next most common.

In some cases the family tendency was remarkably strong; in one, not less than fourteen members, in four generations, being affected.

An equally striking instance is reported by *L. C. Gray*.

The influence of alcohol in exciting to the transmission of epilepsy is verified—though for but a limited number of cases, under treatment in the Salpêtrière—by *Martin*, and the same writer points out that the actual infecting power of the ancestral taint is made less evident by the figures given than it would be, but for the fact that epileptics have but few children, and many of these die young. Similar views are held by *E. C. Mann*, of Hartford.

The following may here be quoted from the second edition of *Nothnagel's* article: "It is still a matter of doubt whether the marriage of near kin serves as a predisposing cause of epilepsy, as a certain number of cases have seemed to show. The feeling has been growing gradually stronger that the harm resulting from such marriages—so far as the influence of the consanguinity per se is concerned—has been overrated. *Henry Huth* takes this ground, and *George Darwin* has come to the conclusion, from careful statistical investigations, that numerical evidence does not support the current belief. If the husband and wife are both of healthy parentage, the fact that they are blood relations has not been shown to entail disease to their descendants." *

Gowers, like *Nothnagel*, has not been led to attach much importance to phthisis as a predisposing cause of epilepsy.

It is but proper, in connection with these statements as to hereditary transmission, to see what light, even of a speculative nature, there is to guide us in determining which the facts are among those offered or to be sought for, which have the most real significance. What lesion, in short, is it which is transmitted? Is the vice of structure or nutrition in the nerve-centres, whose "discharge" causes the epileptic fit, inherited in the very form in which it existed in the parent, or is it some more general disorder of nutrition, centring, perhaps, round the processes of digestion or circulation, which causes epilepsy as it might cause any one of a variety of diseases? It is manifest that this question is an important one, as *Hughlings Jackson* long ago pointed out, for, if the cause or the partial cause of the perverted nutrition which underlies the hyper-irritability of the ganglionic centres is, for instance, of circulatory origin, we are neglecting the direct path of inquiry in not turning our attention to that condition in the patient's ancestry rather than to other diseases of the nervous system, which, however often associated with epilepsy, may be so only in so far as both spring from similar causes.

---

* In spite of this verdict of non-proven, there is much circumstantial evidence for the view that consanguineous marriages may cause both epilepsy and blindness or mutism, and in making a practical decision it is well to err on the safe side.

This is the position taken by *Hughlings Jackson.* The various neuroses mentioned have, as he says, as yet no recognized pathology, and neither the presence of them nor of epilepsy itself in the parent can therefore be taken to prove that epilepsy may be inherited as epilepsy, or as a nervous disease at all. *Jackson's* hypothesis is still without definite support, but is nevertheless not rebutted, and cannot be till we have learned better how to localize the lesions of epilepsy and to examine their nature.

The statistics of *Gowers* are worthy of mention here, as showing in greater detail than has been done before, the influence of age and sex in determining the outbreak of epilepsy, though his figures support the statements of *Nothnagel* and others.

Thus we find that between the ages of nine and sixteen, while puberty is being established, the frequency with which chronic epilepsy begins is on the steady increase, then that it begins to fall off pretty rapidly.

Further, in the case of girls, the figures between twelve and sixteen run up much higher than they do for boys, and this is especially true of the hereditary cases.

It is impossible not to be struck with the similarity of these curves to those given by *H. P. Bowditch* and others, which show the relative rates of growth of boys and girls at this period. In them the line for the girls crosses that for the boys at twelve, and continues to diverge from it till fourteen and a half, when it rapidly approaches it again, though girls remain taller and heavier than boys till over fifteen. It is evident that the increase in the rate of growth is not the sole cause of the preponderance of the epileptic tendency in girls at this period, since the maximum of the latter occurs (*Gowers*) at sixteen, that of the former at fourteen and a half, a discrepancy for which various causes suggest themselves as possible.

In connection with the greater frequency with which epilepsy first shows itself during the period of puberty, the observations of *Lasègue* must be mentioned, according to whom a large proportion of these cases are due to malformations of the cranium, detectable by careful measurement about the face and head, but centring at the base of the skull and associated with abnormal ossification at the sutures near the foramen magnum (comp. the original art. in this Cyclopædia, by *Nothnagel*).

A more extended notice would be given of these observations, but that doubt has been thrown on their importance by the parallel investigations of *Garel,* who shows that this asymmetry is not much more common in epileptics than in healthy persons. *Nothnagel* also emphasizes the fact that patients with epilepsy are often in blooming health and free from deformity.

Among the exciting causes of epilepsy, carefully enumerated and studied by *Gowers*, rhachitis is especially worthy of mention, both because it is, as he thinks, a frequent cause, through the imperfections of nutrition of the nervous system which it entails, and because it is in a measure curable.

The infancy of children with this disease, especially if they inherit likewise an epileptic taint, is to be watched over with extreme care.

SYMPTOMATOLOGY.

Careful observations by *Magnan* as to the condition of the heart, and the blood-pressure, during epileptic attacks in the dog, occurring as a result of absinthe poisoning, have shown that during the period of tonic spasm the blood-pressure rises very high, and the heart beats strongly and rapidly, but with short excursions, with incomplete diastole. During the period of clonic spasm the blood-pressure falls again rapidly, even far below the normal, and the heart's action becomes very slow. That these phenomena were not the result of the convulsion itself was shown by the fact that they did not occur if the vagus had been previously cut.

Among unusual symptoms of epilepsy may be mentioned the liability to fall asleep at unseasonable times, while talking, at table, even while walking in the street. Cases of this kind have been reported by *Westphal* and later by *Fischer*, and I have seen a marked instance of the kind. *Westphal's* case was not, in the ordinary sense of the word, distinctly epileptic nor does he give it that name. Besides the attacks in which the patient actually fell asleep, he had others in which he was only asleep to appearance, retaining some degree of power to hear and understand what was said, and besides this he had seizures which could perhaps better be called cataleptic than epileptic. The case reported by *Fischer* was more evidently epileptic, and the seizures were preceded by either mental confusion or a sense of heaviness and discomfort in the limbs. In the case observed by myself, one well-marked though slight epileptic attack had been seen by the patient's friends, and indirect evidence was found of others. All three cases were of several years' duration.

With regard to the constitution of the urine after epileptic attacks, *Huppert* reiterates his former statements as to the frequency with which albumen and even hyaline casts are found, and that too in the urine of women as well as of men, and under circumstances which show that this is not a result of venous congestion, but of arterial changes of tension. Analogous effects attend the paralytic attacks which occur in progressive paralytic dementia. In many of the transitory apoplectic seizures which occur in the course of progressive paralytic dementia albuminuria is also seen. This is especially true of those attacks which are characterized by elevation of temperature, while there are others, both of a paralytic and epileptiform nature, where it is absent.

Huppert concludes by saying: "This much, at any rate, may be looked upon as certain, the albuminuria is a much more common consequence of (recent) affections of the central nervous system than has been supposed, and even accompanies processes that have usually been characterized as psychical disorders, such as attacks of acute excitement."

Hughlings Jackson has recently described again, in three lectures before the Harveian Society, his views as to the nature of epilepsy and the epileptic attacks, and though they have been stated in similar language on former occasions, yet it has been thought worth while to attempt a brief abstract of them here, because they do not seem to me to have attracted the amount, or still more, the kind of attention that they deserve. This is, no doubt, due in the main to the philosophical style and language in which the reasoning is couched, and to the unusual point of view from which the subject is regarded, which has given his papers the air of a contribution rather to psychology than to medicine, and has caused them to be often misunderstood, and often set aside as "theoretical."

As a matter of fact, it is a pre-requisite to the comprehension of this branch of cerebral physiology and pathology that the physician should be familiar with the elements of psychological reasoning, but, with that granted, the views of *Jackson* seem to me the most lucid, straightforward, simple, and well-grounded that we possess.

The usual belief on the continent of Europe with regard to the pathology of epilepsy, and that held pre-eminently by *Nothnagel*, is that it is an affection, functional or organic, of the pons and medulla oblongata. When we come to study the grounds for this opinion, we find them so scanty that the whole doctrine hardly deserves to be looked on as more than a pure hypothesis, in spite of the very able arguments of *Nothnagel* in its support, and as a hypothesis it seems to me far less direct and simple than that of *Jackson*, which looks upon epilepsy not as being one and the same thing in every case, perhaps not so in any two cases, but as differing according as one or another portion of the ganglionic matter of the brain takes on this peculiar irritability which constitutes the disease.

In order to seek out the portion of the diseased part in any given case, *Jackson* interrogates the attack itself with all its attendant phenomena, and especially the earliest manifestations of the attack, as reflecting, though crudely, the functions of the ganglionic centres which are involved. It is true that the "vaso-motor" doctrine rests on the apparently broad footing of the celebrated experiments of *Kussmaul* and *Tenner*, as well as on the fact that the parts named contain ganglia (Krampf-centrum), the excitation of which is capable of producing generalized muscular convulsions, and vaso-motor centres, which no doubt control the blood-supply of the whole body, and may modify that of one part, as the brain, without the rest. Still it is questionable whether these arguments, drawn from physiological experimentation, important as they are, have not been pushed too far, so as to draw away the attention from other lines of research.

It is a habit with many writers to speak of cases of epilepsy, or so-called epileptiform convulsions, if they are due to irritation of the cortex cerebri, as Jacksonian or cortical epilepsy, but, as a matter of fact, the special merit of *Jackson's* work lies less in his pointing out that the primary lesion of many cases of epilepsy is probably situated in the cortex, than in his indicating the proper method of investigating *all* cases of epilepsy. There seems, certainly, to be no presumptive reason why the convulsions of epilepsy should not, and sometimes may not have their origin in an excitation of the "convulsion-centre" of the pons varolii as well as of other parts, if only it could be shown from the character of the convulsion that this diagnosis was probable. In other words, it is premature to regard epilepsy as a definite disease, always repeating itself, like one of the exanthemata, but all that we have a right to affirm is, that the single common element in different cases of epilepsy is a permanent liability on the part of ganglionic matter somewhere to enter into undue activity, to "discharge," either from causes which if external are slight, or are internal and bound up in the life of the organism. To say where this ganglionic matter is situated is a question for special investigation in each case, though there are unquestionably certain locations, within wide limits, which are affected by predilection.

We should not speak of *epilepsy*, but of *epilepsies*, seeking for differences between them in the character of the auras and the other early manifestations of the attacks, before the picture has become confused by the too great generalization of the symptoms.

So far, it would seem, that we can go safely. When it comes to deciding as to the ultimate cause of this "dischargeability" or irritability of these various nervous centres, we enter into the domain of hypothesis, and certainly the simplest explanation would not be that which considers the vaso-motor centres in the medulla to blame for all the varied forms in which epileptic attacks show themselves. It is far easier to assume, with *Jackson*, that these imperfections of nutrition of the various nervous centres are purely local, leaving it as a matter for future investigation whether their ultimate cause is a congenital vice of development on the part of the tissue-elements, or an imperfection of the local blood supply, or both. But, it will be said, it is comprehensible that these minute localized lesions, situated, no doubt, oftenest in the cortex cerebri, should cause localized symptoms, such as muscular spasms, but not that they should cause all the phenomena of a full epileptic attack. Nor is this supposed to be the case. It is only maintained that the epileptic discharge *begins* in these circumscribed portions of ganglionic matter, spreading thence both "laterally and downwards" (in a physiological sense) through the other cerebral centres; just as when we make a strong effort to grasp something with the hand, the innervation spreads gradually from the nervous centres concerned chiefly with the hand to those of the forearm, of the arm, the shoulder, the neck, the face, and the arm of the opposite side of the body, even involving sets of muscles whose contraction adds nothing to the efficiency of the original and still central effort. Even the occurrence of disturbances of the animal or organic functions of the body, hallucinations of smell, sight, hearing, pallor of face (spasm of arteries), salivation, etc., are well explained on this hypothesis, both through psychological reasoning and through experimental evidence, since there has been no more striking result of the recent investigations into the physiology of the cortex cerebri than the discovery that its functions are closely related to those of the parts concerned in organic and vegetative life, the heart, the blood-vessels, the glands, etc.

In the recent papers alluded to, the author devotes himself mainly to the study of the seizures of what is clinically known as epilepsy proper, characterized by early loss of consciousness, rapidly and widely spreading spasm, etc., setting aside the so-called epileptiform convulsions (monospasms, or what is sometimes described as cortical or partial epilepsy), in which consciousness is lost late if at all, and the muscular convulsion spreads less rapidly and less widely, not as belonging scientifically in a different genus, but for reasons of convenience.

Jackson shows that, in these cases of epilepsy proper, the "discharge" which causes the fit takes place, not in the ganglionic matter of the so-called "motor region of *Hitzig* and *Ferrier*," in which the discharge causing the epileptiform convulsions originates, but in other parts of the cortex, where nervous arrangements exist which serve as the anatomical substratum of the higher mental operations, but points out that the significance of the complex features of these seizures is hardly to be understood till the simpler "partial" attacks have been studied.

The difficult task of summarizing *Jackson's* searching analyses will not be attempted here. The reader is referred to his various papers, as well as to the recent lectures of *Gowers*, by whom the opinions of *Jackson* are in the main adopted.

I must also abstain, for want of space, from discussing at length the important question as to what we are to understand by "post-epileptic" states.

PATHOLOGICAL ANATOMY.

Hemkes, and more recently *Pfleger*, have reported quite a number of instances of the mysterious sclerosis of the cornu ammonis; the latter twenty-five times out of forty-three cases, the former six times out of thirty-four cases. Neither writer looks upon this as a primary lesion, but either as secondary, or else as indicating generalized cerebral disease.

Still more lately, *Sommer* has given the results of an exhaustive study of this subject in a monograph (*Arch. für Psych.*, etc., X., 3), in which he has collected and analyzed ninety records of autopsies in which some change of the Ammon's horn was found, besides describing an accurate topographial and histological investigation in a case of his own. He believes that the relation between the lesion and the epilepsy is far more than accidental or secondary, the former occurring, as it does, in about thirty per cent of all fatal cases, and he thinks that the sensory auras which are so common may originate in disease of the part in question, to which sensory functions have been attributed by *Ferrier*. The theory is suggestive, but at present hardly more, and the fact that the cornu ammonis of both sides is usually involved seems to me to militate against it.

The microscopic changes found in *Sommer's* case consisted essentially in a disintegration and disappearance of ganglion-cells, with degeneration of the adjacent fibrous tracts.

Gowers thinks, and I cannot but agree with him, that, from the physiological stand-point, the nature of the "epileptic condition" of ganglionic matter is to be sought, not in "increased irritability" or "hypernutrition," but in the loss of the inhibitory function of the nerve-cell. The researches of the physiologists (*Wundt*) certainly lead to the belief that tension or self-inhibition is a function of all ganglionic matter which is first to suffer when the nutrition is impaired.

TREATMENT.

Whatever be the views, anatomical or physiological, which each may hold as to the nature of the change in the nervous centres which underlies the epileptic state, nobody will object to the proposition that it is one with which a local imperfection of nutrition somewhere has much to do. Attempts to cope with this condition have followed mainly two lines, the dietetic and the sedative. The most successful attempt of the former kind has been by the disuse of animal food, which is still a cherished means of treatment (compare especially *Merson*, West-Riding Asylum Reports, Vol. V., 1875). *Lépine* has reported a carefully studied case, of much therapeutic interest, where both these methods were successfully combined.

The patient was a plethoric man and the attacks had been recurring with great frequency, four or five times each night.

The treatment consisted in the use of a bland and very restricted diet, the employment of frequent venesections, by which the globules of the blood were reduced by nearly two millions to the cubic centimetre, and the administration of large doses of digitalis and bromide of potassium combined, the latter alone failing to accomplish the desired end, and both failing to do so if unassociated with the bleeding. At the end of forty-four days after this treatment had been got fairly under way, the patient had had no attack. It would not do, as *Lépine* says, to treat all cases of epilepsy with bleeding and depletion, the more so that it was formerly a favorite means of treatment in the French hospitals and has since fallen into disuse, presumably because too indiscriminately employed. At the same time, it cannot be assumed too hastily that it is only in "plethoric" cases that this method is applicable. That would be to take for granted a greater similarity between the local and the general condition than is justified by our present knowledge. No doubt, however, these would be the best cases to begin the investigation upon.

There are, similarly, classes of cases, as *Gowers* has shown, where iron is distinctly useful, others where it seems injurious, though we cannot always recognize them in advance.

With regard to the effects of trephining in traumatic epilepsy, new instances of success are reported from time to time.

Echeverria gives a table of one hundred and forty-five cases of trephining for traumatic epilepsy, claiming to be all reported up to 1878. The question as to the value of this treatment is not one that admits of solution by statistical investigation, the cases are so utterly different; but that good effects sometimes followed is indisputable, even where the operation was done as late as ten, fifteen, even twenty years after the injury, and even, what is more remarkable, where no affection of the inner table was found.

J. F. West has recently reported a case of this latter kind and suggests that the irritation causing the epilepsy may have originated in the lesions of the outer layers of the bone, referring to a case following necrosis of the tibia, where operation gave relief.

E. C. Seguin reports observations showing the uselessness of chloride of potassium as compared with the bromine salts in controlling the paroxysms, and on the value of the combination of bromide of potassium with chloral hydrate, both in moderate doses, as, at any rate, often making it possible to dispense with the large and sometimes objectionable doses of bromide.

Giersing studied with great care the comparative value of a number of drugs, in three cases, occurring in one family, and followed for a series of years. With each drug the dose was gradually increased till it seemed to have reached the limit of its usefulness. The main results were as follows:

Extr. of cal. bean,	given during 862 days.	Attacks every 9.5 day.
Brom. of potass.	" " 444 "	" " 18.5 "
" " with bellad. or atrop.,	" 2,390 "	" 36.2 "
" " with digitalis,	986 "	" " 23.6 "
" " " valerian,	329 "	" " 54.9 "

Of other new remedies tried often enough to make statements about them of suggestive value, I will mention, subcutaneous injections of curare, used by *Kunze* in eighty cases. The mixture employed was:

Curare	0.3
Aq. dest.	5.0
Mucilag.	gtt. 2

Of this, eight drops were injected every fifth to seventh day for several weeks at a time, alcoholic drinks being meantime avoided, and, in obstinate cases, milk diet enforced.

Six cases are reported as having been radically cured.

The first signs of toxic effect were slight amblyopia and mental confusion. (*K.'s* original papers I have not been able to see.)

Sometimes the systematic aborting of epileptic attacks is capable of exerting a permanent influence upon the epileptic condition. This is shown in a case referred to by *Gowers*, where the fits were checked by the old method of tightening a ligature round the arm, with the effect finally, that the auras always ceased of themselves at the seat of the ligature, and no fits followed.

Another successful method of aborting attacks is by the subcutaneous injection of apomorphine (*Vallender*), and—what is more important, because oftener practicable—the ingestion of a handful of common salt *Nothnagel, Schulz*). The effect of these agents is, no doubt, produced through their inhibitory action, and it is very interesting, as *Gowers* points out, to see that a condition of permanent inhibition may at times be thus set up, the process underlying the aura learning, as it were, to call into existence through frequent association, the counter process, by which, when artificially excited, it had itself so often been checked. Unfortunately, it is only very exceptionally that we are in a position to use these remedies with the necessary promptness and regularity; the salt, however, more so perhaps than any other.

One patient of my own believed himself able to check his attacks in this way.

The remarks by *Gowers* upon treatment, in the Gulstonian lectures upon epilepsy above referred to, are worthy of mention, though less for their statistical value—his notes covering but 562 cases—than on account of the evident care with which the observations were made. The only new remedy that is brought forward is borax, and since in this distressing malady any treatment that can be used without danger is especially welcome, I quote the paragraph concerning this, entire: "In several inveterate cases in which bromide had no effect, I have tried borax. In some cases it did no good, but in twelve its value was most distinct. I may mention one or two. In one, fits which had continued on bromide and on zinc, ceased entirely on borax for three months, and then only recurred when the medicine was discontinued. In another case, the fits continued, about one weekly, during three months' treatment on bromide and on belladonna. Borax was then substituted; the fits at once ceased, and for five months the patient had not a single fit; then he had one in each of the two following months; the dose of borax was increased, and up to the present time, eight months later, no other attack has occurred.

"In a third case, one or two attacks occurred once a fortnight, on bromide. Borax was substituted, and for five months the patient had not a single fit.

"The doses given have been ten or fifteen grains twice or three times a day.

"It produces in some patients gastro-intestinal disturbance. and. rarely, a form of dysenteric diarrhœa.

"By others it is well borne, and one of my patients has taken forty-five grains a day for twelve months without the slightest inconvenience, and says that no medicine has ever done him so much good. In cases in which bromide fails, borax certainly deserves a trial."

Some interesting experiments by Dr. *Ramskill* upon the action of picrotoxine on four epileptic patients are quoted, by which it appears that the subcutaneous injection of fifteen to eighteen milligrammes almost invariably excited a severe fit, usually preceded by its regular attendant aura. Neither in these nor in seven other cases in which smaller doses (one to four milligr. daily) were employed, was any favorable therapeutic effect found to attend its use.

Dr. *Gowers*, also, had used picrotoxine in a few instances, but only in one had its action been found to be beneficial. On the other hand, *Planat*, *Hambursin*, *Couyba* speak with much favor of the effects of the drug, though the number of cases reported is but few.

Hambursin (*Bull. de l'Acad. de méd. de Belgique*, 1880. Abstr. in the *Jr. de méd. et chir. pratiques*, 1880, p. 177) prescribes the alcoholic tincture of Cocculus Indicus in the dose of ten drops, to be increased gradually, day by day, to one hundred, or even one hundred and fifty; and thinks that *Planat's* maximum dose of sixty drops is often too small, since the system soon becomes habituated to the remedy and a larger dose is required to produce a given effect. It can be used in conjunction with bromide of potassium, if desired. His report covers six cases.

Couyba (*Jr. de méd. et de chir. prats.*, 1880, 214) describes with some detail the case of a child five years of age, in which, as long ago as in 1877, he used picrotoxine with great benefit, basing the treatment on the physiological action of the poison as pointed out by *Gubler*.

The patient had been epileptic since his second year; and for the past year the attacks had recurred regularly once a month. Bromide of potassium, in doses of three grammes a day, had failed to influence them.

On the 6th of January, 1877, the picrotoxine treatment was begun, 0.0005 being given in pill, at first twice daily, then three times, as the period of the February attack approached. The largest dose at any time was five pills daily, and from time to time the treatment was entirely suspended. During 1877 there were but three full attacks, and three of migraine or vertigo in place of others. During 1878 there were two full attacks and four of vertigo; during 1879 one attack of vertigo; during the first four months of 1880 (date of writing), no attacks of any kind.

The conclusions of *Gowers* with regard to the alkaline bromides do not differ essentially from those which command the acceptance of most observers, viz., that, either alone or in combination, they usually relieve more or less, and occasionally cure. The most active combination was found to be that with digitalis, and this, too. even when no cardiac symptoms were present.

Reasoning from the assumption that the bromides owe their efficacy to their increasing the molecular stability of the nerve-cells, by inducing some nutritive change in them, Dr. *Gowers* concluded that there is no reason to limit their dose to the amounts necessary to check the fits, and has tried giving much larger quantities every two or three days, increased gradually even up to an ounce at a single dose.

This, he says, produces in some patients headache only; in others, slight stupor, reaching its maximum on the second day. On account of the varying susceptibility of different persons, however, he recommends not beginning this method of treatment with a larger maximum dose than half an ounce. Even this dose, one would think, should never be used except under the physician's very eye.

It is unfortunate that there exists so much difference of opinion with regard to such points as the effect of combining the so-called tonics, or stimulants, such as quinine, iron, strychnine, with the bromides, or as to their action in epilepsy when given alone (see for instance a discussion in the American Neurological Association, apropos of a paper by Dr. *L. C. Gray*, speaking very favorably of the use of quinine with the bromides, published in the Chicago *Journ. of Nervous and Mental Diseases*, for July, 1880), or again as to the effect of a low, or non-animal diet.

It may be that a portion of the disagreement is due to the fact that the various observers have dealt with different classes of cases (see *Lépine's* case, above), although on this point we cannot speak with certainty.

TETANUS.

BIBLIOGRAPHY.

CHOPARD, A.: Contrib. à l'étude du tétan. Paris, 1876 (contains numerous references).—BARUCH, S.: Report of the Committee on Tetanus. Tr. S. Car. M. Ass., Charleston, 1877, 79–83.—BEARD, G. M.: The Endemic Tetanus of Eastern Long Island. Tr. Am. Neurol. Ass., N. Y., 1877, II., 50–75.—TAYLOR, F.: Report on Cases of Tetanus. Guy's Hosp. Rep., London, 1878, XXIII., 339–385.—WETMORE, C. H.: On Tetanus in the Hawaiian Islands. M. and S. Reporter, Phila., 1878, XXXIX., 117-119.—YANDELL, D. W.: A Study of Four Hundred and Fifteen Cases of Tetanus. Brain J. Neurol., London, 1878, I., 340-347.—RATTON, J. J. L.: Brain, V., 2, No. 4, 1879. On the Origin of Tetanus.—TYSON, J.: The Results of a Minute Examination of the Spinal Cord in Two Cases of Acute Tetanus. Practitioner, Lond., 1877, XIX., 109–114.—LAVERAN, A.: Contribution à l'anatomie pathologique de tétanos et de la névrite ascendante aiguë. Arch. de physiol. norm. et path., Par., 1877, IV., 695–705.—RINGER, S. and MURRELL, W.: Further Observations and Experiments Regarding the True Nature of Tetanus. Jour. Anat. and Phys., Lond., 1877, XI., 517–528.—AUFRECHT, E.: Zur pathologischen Anatomie des Rückenmarks beim Tetanus. Deutsche med. Wochenschr., Berl., 1878, IV., 160–192.—COATS, J.: On the Pathology of Tetanus and Hydrophobia. Med. Chir. Tr., Lond., 1878, LXI., 79–94, 2 pl.—TRAUBE, L.: Ein paar Bemerkungen über Tetanus. Ges. Beitr. z. Path. u. Physiol., Berl., 1878, III., 561–562.—WOODS, G. A.: A Contribution to the Pathology of Tetanus or Lockjaw. Lancet, Lond., 1878, II., 326–328.—ROSS, J.: The Pathology of Tetanus. Med.

Times and Gaz., Lond., 1878, II., 527.—JACKSON, HUGHLINGS: Remarks on the Comparison and Contrast betwixt Tetanus and a certain Epileptiform Seizure. Med. Times and Gaz., Lond., 1878, II., 484–485.—FOX, E. L.: Contribution to the Pathology of Tetanus. Tr. Bristol Med. Chir. Soc., 1878, I., 60-64.—CARRINGTON, R. E. and WRIGHT, G. A.: Note of a Case of Tetanus in which Extensive Lesions were found in the Spinal Cord. Guy's Hosp. Rep., Lond., 1879, 3 s., XXIV., 185–190.—AMIDON, R. W.: Some New Points on the Pathological Anatomy of Tetanus. Arch. of Med., N. Y., 1879, I., 265–282, 1 pl.—ROSS: Trans. of the Path. Soc. of London, 1879, p. 215.—DORAN, HARRIS: Lond. Lancet, April 10th, 1880.—WAGSTAFFE: A Case of Traumatic Tetanus Treated by Profuse Sweating, Cured. Brit. M. J., Lond., 1877, II., 563.—On the State of Therapeutics in Tetanus. Practitioner, London, 1877, XIX., 115-121.—THORPE, J. C.: A Case of Traumatic Tetanus Treated with Large Doses of Tincture of Aconite. Recovery. J. Neu. and Ment. Dis., Chicago, 1877, IV., 297–300.—SPRATLEY: Two Cases of Trismus Successfully Treated with Gelseminum. Liverpool and Manchester M. and S. Rep., 1877.—OWENS, J. E.: Lacerated Wound of Fore-Arm; Treatment by Calabar Bean; Stretching of Brachial Plexus; Death. Chicago Med. Jour. and Exam., 1877, XXXV., 599-604.—NANKIVELL: Two Cases of Traumatic Tetanus Treated by Stretching of the Median Nerve; Death in both Instances. Lancet, Lond., 1878, I., 311–312.—CALLENDER, G. W.: Nerve-Stretching in Tetanus· St. Barth. Hosp. Rep., Lond., 1878, XIV., 193–195.—FENN, C. M.: Nerve-Section in Traumatic Tetanus. Am. M. Bi-weekly, Louisville, 1878, VIII., 217–218.—KLAMROTH: Fall von Tetanus traumaticus, erfolglos mit Nervendehnung behandelt. Deutsche med. Wochenschr., Berl., 1878, IV., 546–547.—MORRIS, H.: A Case of Acute Tetanus Treated by Stretching the Great Sciatic Nerve. Brit. M. J., Lond., 1879, I., 933.—MORRIS, H.: Nerve Stretching in Tetanus. Lancet, Lond., 1879, II., 963.—JOHNSTONE, A. W.: Nerve Stretching in Tetanus. Lancet, Lond., 1879, II., 892.—KLIN, E. and KNIE, A.: Casuistischer Beitrag zur Behandlung des Tetanus traumaticus mit Nervendehnung. St. Petersb. med. Wochenschr., 1879, IV., 307.—THOMAS, L.: Tétanos consécutif à une plaie de la main; élongation du nerf médian; cessation de la contracture et des spasmes; mort par infection purulente. Bull. et mém. Soc. de chir. de Par., 1879, n. s., V., 173–176.—HUTCHINSON: Nerve-Stretching in Tetanus. Rep. by Mr. Wilson. Med. Times and Gaz., Lond., 1879, I., 618.—CLARK, H. E.: A Case of Tetanus Treated by Nerve Stretching. Glasgow M. J., 1879, XII., 11–17.—RANSOHOFF, J.: Tetanus; Nerve-Stretching; Cure. Cincin. Lancet and Clinic., 1879, n. s., II., 41–43. [For further references to nerve-stretching in tetanus, see the C.bl. für klin. Chir., 1879-80.]

ETIOLOGY AND SYMPTOMATOLOGY.

Dr. *G. M. Beard* has investigated the tetanus of Suffolk Co., Long Island, New York, where both the spontaneous and traumatic form are endemic, and concludes that it is probably due to the dampness of the ocean air, combined with local dampness of the soil. It has been on the decrease for the past ten or fifteen years.

Dr. *Yandell*, from the analysis of 415 cases, comes to conclusions not essentially differing from those of other observers. He finds, in accord with Dr. *Taylor*, who gives an analysis of 51 cases treated at Guy's Hospital, that the length of the period supervening between the injury and the outbreak of the symptoms is an important element in the prognosis, "the largest number of recoveries being found in cases in which the disease occurred after the lapse of nine days from the injury."

Of *Taylor's* cases, those due to the slighter injuries ran on the whole the milder course.

According to *Yandell*, the disease is more fatal if it occurs during pregnancy.

PATHOLOGICAL ANATOMY.

Quite a number of cases have been reported in which positive changes have been found in the central nervous system of persons dying with tetanus. The significance of these changes is, however, a matter of doubt. Thus *Coats* reports the examination of five cases occurring in man, and one in the horse, where changes were present analogous to those described by earlier writers, viz., 1, great overfilling of vessels, occurring irregularly; 2, granular exudations around the vessels, with occasionally small hemorrhages, etc. These changes were most marked in the medulla, but present to a less degree in the pons, cerebral ganglia, and even in the convolutions. "Gaps" in the tissues are spoken of, and are believed to be due to the falling out of vessels with the exudation matter surrounding them. In the accounts of other writers, similar spaces (vacuoles, etc.) are described and even figured. It is questionable, however, whether such spaces, whether empty or filled with "colloid" material, may not be the result of shrinkage during hardening. The absence of distinct signs of inflammation in their vicinity makes it improbable that they are of myelitic origin. The convolutions were examined in two cases, and a yellowish material seen surrounding the blood-vessels there.

The nerve coming from the injured part was examined in one case and found healthy

Aufrecht found intense and wide-spread pigmentary degeneration ("parenchymatous inflammation") of the ganglion-cells in the lumbar and cervical enlargement of a patient who died on the third day after the outbreak of symptoms. In the cervical and upper dorsal region were also numerous globular bodies of fatty appearance, perhaps similar to those spoken of by *Coats* and *Quinquaud* as occurring in the neighborhood of blood-vessels. *Woods* found great vascular engorgement, especially in the neighborhood of the hypoglossal and pneumogastric nuclei, also in the spinal cord, especially around the central canal, and the portion nearest the seat of injury.

In the same neighborhood there was a diffused infiltration of the parts with leucocytes, and granular disintegration, especially in the posterior columns.

The author regards these changes as being secondary to an increased reflex excitability of the spinal centres, which he thinks may arise from various causes. *Laveran* found diffused interstitial disease of the cord, as well as of a limited segment of the nerve coming from the injured part, through its whole length, without change in the ganglion-cells. The case was one of severe crushing of one leg, causing death on the fourteenth day.

In the case described by *Carrington* and *Wright*, of a patient who died on the sixth day after his injury (severe burns of the leg) and *eight* *hours* after the outbreak of symptoms, the right half of the cord, in the cervical region, was distended laterally, and after hardening, a cavity was found running the whole length of the cervical enlargement, in the right cornu, and a similar but smaller cavity in the left half of the lumbar enlargement. The vessels everywhere were engorged, and the tissues immediately adjacent to the cavities somewhat altered, but no other marked signs of disease were present, except thickening of the pia mater in the diseased region.

Amidon has studied the topographical distribution of the lesions in one case with great care, though the changes themselves—which varied from vascular engorgement to localized disintegration and vacuole formation—are not essentially different from those which have been ascribed by other writers to congestion and post-mortem influences. The important point is, that they were found mainly confined to the tracts occupied by the trigeminal, part of the facial, spinal accessory, and hypoglossal, none of these parts being, however, entirely destroyed. The glosso-pharyngeal and pneumogastric tracts were but little involved. For the interpretation of the clinical phenomena, in the light of these lesions, the reader is referred to the original paper.

It is evident that accurate topographical researches of this kind may give a significant value to alterations of structure not pathognomonic in themselves.

Ross has recently described lesions which differ from those of other writers, in that the ganglion-cells in certain locations, throughout nearly the whole length of the cord, are said to have either disappeared or become greatly shrunken. These cells seem to have occupied rather the central portions of the anterior columns, being the outlying cells of the principal groups. The vesicular column of *Clark* is also said to have suffered, especially in the lower cervical and upper dorsal region. There was everywhere a considerable but diffused infiltration of leucocytes. In the medulla, some of the anterior and internal cells of the hypoglossal nucleus were affected, while the body of the nucleus was healthy, and the nuclei of the pneumogastric and spinal accessory were also involved.

These observations, in view of the negative results of other examinations, are not be received without careful criticism. In two cases studied recently by *Doran* and *Harris*, no such shrinking of the cells, as is spoken of by *Ross*, was found, nor any other important changes. A few of the transparent spaces filled with clear material, noted by *Coats* and others, were seen.

The same doubts are pertinent to-day that were raised by *Schultze* in 1877, whether these various changes are to be looked upon as the causes or as the concomitants and results of the real lesion, whatever that may be. Congestion is met with in other organs besides the cord, and exudation is a common consequence of congestion. Even vacuoles of considerable size have been found in perfectly healthy cords, and still more in those which had been the seat of vascular engorgement and exudation, as the result of changes occurring *post mortem* during hardening.

It appears certain, at least, that in some well-authenticated cases no changes of note have been found either in the nerves of the affected part, or in the central organs.

These criticisms would be in a measure disposed of if it could be shown that the lesions found in tetanus, even if slight and not pathognomonic, predominated in those central tracts which corresponded to the affected groups of muscles. The lesions are at any rate not so definite and uniform, either in character or location, as those which have been found in the more recent cases of hydrophobia by *Gowers* and several others, and are, therefore, more likely to be secondary to the functional disturbance of the nerve-centres than the cause of the latter.

It is easy to conceive, with *Woods*, that the excessive vascular engorgement, when present, should influence the nutrition of the ganglion-cells, but less easy to explain the engorgement, either by calling in the aid of peripheral nerve-injuries, or by referring it to increased excitability of the nervous centres.

TREATMENT.

It has become evident that no "specific" is likely to be found for tetanus. This fact should be recognized, as Dr. *H. C. Wood* points out, and our efforts directed towards meeting the symptomatic indications of each case by a judicious combination of remedies. These indications are, to ward off exhaustion, by giving nourishing food at short intervals (eggs and milk, scraped raw meat, brandy), and to prevent the convulsions from becoming a source of danger in themselves. In deciding upon doses of the various narcotics, it is to be remembered, *Wood* says, that nervous centres are less susceptible to them than in health. "Potassic bromide may be given up to gms. 30.0 daily without danger, and chloral may be risked in doses of gms. 2.50 to 3.00 if necessary." *Wood* prefers to keep opium and chloral for use at night, relying mainly on potassic bromide through the day, and using calabar bean and curare as adjuncts.

It is to be remembered that (according to *Yandell's* conclusions from the analysis of 415 cases) "when tetanus continues fourteen days, recovery is the rule, and death the exception, apparently independent of the treatment." Chloroform is often of great service. The doses of calabar bean sometimes given are enormous, up to gms. 5.50 of the alcoholic extract in one day, in a case reported by *Read*. Nerve-stretching as a cure has not fulfilled what was hoped of it, and in some cases the operation has even been followed by increased violence of the spasms (*vide* Bibliograph. Index.) This treatment, however, still finds warm adherents among distinguished surgeons, and as it has often seemed to be of service, it is well worth trying, especially where surgical interference is called for, either in connection with nerve section, or in place of it, as being the milder operation.

It has been recommended to stretch the nerve as near as possible to the spinal cord, and by a series of quick, forcible pulls. There seems no doubt that in this way the reflex excitability of the cord can be lowered (v. *Bost. Med. and Surg. Jour.*, August 24th, 1880).

PARALYSIS AGITANS.

BIBLIOGRAPHY.

RABOT: Lyon Méd., 1874, Bd. 22.—HUCHARD: Union méd., 1875, 7.—SCHULTZE, F.: Ueber das Verhältniss der Paralysis agitans zur multiplen Sklerose des Rückenmarks. Virchow's Arch., Bd. 68, Heft 1, S. 120.—WESTPHAL: Paralysis agitans. Vorwiegende Betheiligung der linken Extremitäten, des Kopfes, Unterkiefers und der Mundmusculatur; Autopsie. Negativer Befund. Charité Ann., Berl., 1876, III., 361–371.—BOUCHER, A.: De la Maladie de Parkinson (Paralysie agitante) et en particulier de la forme fruste. 4to, Paris, 1877 [Thèse No. 76].—HARDY: Paralysie agitante à forme fruste. Gaz. d. Hôp., Par., 1877, L. 913-914.—CHÉRON, J.: De la Modification importante que subit la constitution chimique de l'Urine dans la Paralysie agitante (Phosphaturie). Progrès méd., Par., 1877, V., 903-905. —MARAGLIANO, E.: Sulla paralise agitante. [Ext. Gior. ven. de Sc. méd., 1877, XXVII., 1878, XXVIII.] In his: Lezioni di clin. med. 8vo, Venezia, 1878, 60–105.—DEBOVE: Note sur un cas de latéropulsion oculaire dans la paralysie agitante. Progrés méd., Par., 1878, VI., 116–117.—CHARCOT: Par. agit. à forme fruste. Gaz. des Hôp., No. 2, 1878.—GRASSET, J., et APOLINARIO, B.: Note sur l'état de la témpérature périphérique dans un cas de paralysie agitante et sur l'Influence des Contractions musculaires sur la température periphérique normale. Progrès méd, Par., 1878, VI., 216–217.—HERTERICH, M.: Zur Lehre der Paral. agitans. Würzburg, 1878.—BAUER, J.: Zwei Fälle von Paralysis agitans mit tödtlichem Ausgange. Ann. d. städt. allg. Krankh. zu München, 1878, I., 134–140.—DEMANGE: Rév. Méd. de l'Est., Oct. 15th, 1879 (abstract in the Révue des Sc. Méd.).

SYMPTOMATOLOGY.

Several of the French observers (*Charcot*, *Boucher*, *Hardy*, *Brochin*, *Bourneville*) have done good service in calling attention to the partial, abortive forms (formes frustes) in which this affection sometimes appears.

Charcot has already shown (in his Mal. du syst. nerv.) that of the four or five characteristic symptoms—trembling, rigidity, paresis, altered expression of face and carriage of body—the rigidity, though usually of late occurrence, may come on at the very outset, and it now appears that the trembling, which usually ushers in the case, may be long delayed, or only present from time to time, even though the other symptoms are recognizable beyond question.

Boucher is inclined, on the whole, to regard the general look and carriage of the patient as the most significant sign. *Hardy* confirms the statement of *Charcot* that the oscillation of the head is, in all but rare cases, only apparent, being in reality compensatory to, or communicated by the movements of other parts of the body. *Westphal*, on the other hand, thinks this is not such a very rare symptom, and reports several cases where it was present beyond dispute.

In one of *Westphal's* cases, the head was habitually carried thrown somewhat backwards, instead of forwards, as is the rule.

Another curious symptom is that observed by *Debove*, consisting in an impairment of the lateral motion of the eyeballs. This caused especial annoyance in reading, the patient finding great difficulty in bringing his eyes from the end of one line to the beginning of the line below. In another of *Westphal's* cases the trembling came on four years after the occurrence of cerebral hemiplegia, and affected, first, the paretic hand, then the other. The involuntary movements were, however, persistently slower on the paretic side, where there was also some rigidity, but no notable atrophy. The sensibility was slightly diminished, and was reported to have been more so previously.

The cause of the subjective sense of warmth so often complained of by these patients has been studied by *Grasset* and *Apolinario*, and is ascribed to an actual increase of the superficial heat of the body, which, in one case, was found to be 36.8 (C.), against 33.6 (C.), which they found to be about the average surface temperature in health, taken by the same method.

Chéron has studied the character of the urine in eight cases, though in two only for any considerable length of time. He found the total quantity greatly increased, and the excretion of phosphates augmented even up to three times the normal, and thinks that these changes, together with muscular feebleness, may often be recognized considerably earlier than the trembling.

PATHOLOGICAL ANATOMY.

The recent post-mortem examinations show the same discrepancies with those of earlier date.

In one case observed by *Herterich* (known to me only through an analysis by *Eisenlohr*, in the *C. bl. für Nervenheilkunde*, etc.) in which the symptoms followed typhoid fever—though only after an interval of some years, during which the patient had suffered from weakness and impaired motion of all the extremities—a disseminated sclerosis of the spinal cord was found, recalling the observations of *Cayley*, *Bourillon*, *Schultze*, and others. There was also an extensive patch of degeneration in the floor of the fourth ventricle, reaching forward even as far as the left crus cerebri. There was, unfortunately, no microscopic examination made. The trembling had begun in the lower extremities, but otherwise the symptoms seem to have been characteristic.

In *Schultze's* case, which was carefully observed and critically discussed, the symptoms were still more typical, and the post-mortem changes analogous, in so far as that there were multiple patches of sclerosis present in both.

It is impossible to say in what relation these lesions stood to the continuous muscular tremor in the cases referred to, any more than we can tell in what relation they stand to the characteristic movements of the cases known clinically as mutiple sclerosis, with which they are habitually associated. All that we can say is, as *Schultze* points out, that the absence of the symptoms usually seen in the latter affection, and the presence of those met with in typical cases of paralysis agitans, does not justify us in excluding the pathological diagnosis of "multiple sclerosis." It is improbable that, from the clinical stand-point, these two affections are really akin to each other, yet, in these cases the symptoms of the multiple sclerosis seem to have been either replaced or masked by those of the paralysis agitans, and, inasmuch as both disorders are capable of appearing in such incomplete (abortive) and anomalous forms, it is easy to imagine that the difficulties in the way of a differential diagnosis might occasionally be considerable.

In two cases observed by *Bauer* (also known to me only through a brief reference in the *C. bl. für med. Wissensch.*), the mass of the brain as a whole was found diminished, and in one the medulla oblongata and upper part of the spinal cord notably shrunken.

In a case of *Westphal's* the post-mortem investigation, macroscopic and microscopic, gave entirely negative results, and this, as a matter of fact, *Westphal*, in common with most observers, believes will prove to be the rule in typical, uncomplicated cases of the disease.

Certainly such positive results as have been reported have brought us no nearer to a physiological comprehension of the disease.

In view of the negative post-mortem results in cases examined by such careful observers as *Westphal*, the theory of the disease advanced by *Demange* loses in weight. This author found, on microscopic examination of the cord in a typical case of paralysis agitans, which had been under observation for several years, very slightly-marked sclerotic changes here and there in the antero-lateral columns, together with peri-ependymitis, and—what he regards as of more importance—slight changes in the sensitive tracts, viz., posterior nerve-roots, columns of *Goll*, vesicular columns of *Clarke*. *Demange* believes that the characteristic movements are the reflex result of irritative processes going on in the sphere of the posterior nerve-roots, at first perhaps functional in character, but eventually inflammatory, though rarely becoming sufficiently developed to cause anæsthesia.

Westphal's case was, however, of six years' duration, yet no changes were found.

To show that cerebral activity, while it may control, does not cause these movements, *Demange* reports also an interesting case where hemiplegia came on during the course of paralysis agitans. For several days the patient was unable to use the muscles of the affected side, but the trembling continued undiminished.

TREATMENT.

Of new measures the most important is nerve-stretching, which has been done once in a case of *Westphal's*, with, however, only temporary benefit.

The patient was a man of fifty-eight, and the trembling had come on apparently in consequence of a severe burn. All the large nerve-trunks of the left arm, the part mainly affected, were laid bare just below the axilla and stretched both peripherally and centrally by *Bardeleben*.

The trembling recurred slightly from time to time during the first ten days after the operation, but grew, on the whole, less and less. On the eleventh day, however, the irritation due to a subcutaneous injection of morphia brought back the former condition of affairs. *Westphal* remarks that in view of even this temporary result, and of the intractable nature of the affection, further attempts of the kind are justifiable, but justly calls attention to the fact that we have not yet sounded all the dangers of forcible nerve-stretching, and that he has observed a case where such treatment, applied to the crural nerve, was apparently the cause of a circumscribed myelitis, due to the dragging upon some of the nerve-roots.

Seguin has been able to cause temporary, but only temporary, cessation of the trembling, by the use of hyoscyamia, especially if given subcutaneously. The dose of the crystallized alkaloid is 0.0001 to 0.0002. I have seen striking effects of a similar kind from the use of the fluid extract in one case.

ATHETOSIS.

BIBLIOGRAPHY.

Berger, O.: Berl. kl. Wochenschr., 1877, XIV., 31-45.—Lauenstein: Deutsch. Arch. f. klin. Med., 1877, XX., 158.—Ewald: Dementia paral. Athetosis; Corticaler Erweichungsherd. Deutsch. Arch., 1877, XX., 591.—Ringer: Case of Athetosis Accomp. by Unilateral Sweating. Practit., Lond., 1877, XIX., 9; and 1879, 161. —Jewell: Tr. Am. Neur. Ass., N. Y., 1877, II., 213.—Goldstein: 8vo, Berlin, 1877.—Seguin, E. C.: Tr. Am. Neur. Ass., N. Y., 1877, II., 92.—Lange: Hosp. Tid., R. 2, Bd. 4, 1878.—Oulmont: (Thèse No. 8) Paris, 1878, 4to; also Rev. mens. de méd. et de chir., Par., 1878, II., 81-94.—Dreschfeld: Ibid., 766.—Landouzy: Bull. soc. anat. de Par., 1878, LIII., 15, 549; also Progr. méd., 1878, VI., 79-96 (autopsy).—Kuessner: Athet. Beweg. bei einem Paralytiker ohne Herderkrankung im Gehirn. Arch. f. Psych., 1878. VIII., 443.—Janeway: Post-Paral. Chorea. Hosp. Gaz., N. Y., 1878, 366.—Gnauck: Ueb. primitive Athetose. Arch. f. Psych., 1879, IX., 300.—Erb: Ibid., 277.—Beach, F.: Athet. of Both Sides of the Body, not Assoc. with Epilepsy. Br. Med. Jr., 1879, II., 815.

SYMPTOMATOLOGY.

It appears to be now universally conceded that whatever be the cause of the characteristic symptoms of athetosis, the affection is closely akin to several other forms of mobile spasm, and possibly also of continuous spasm, which follow hemiplegia of cerebral origin.

The analysis of these different forms of cerebral hemi-spasm which is given by *Gowers*, and of which the results are summed up in the following table, is well calculated to bring out the relationship between them :

POST-HEMIPLEGIC DISORDERS OF MOVEMENT.

Quick, clonic spasms, of intermitting type.	Regular (continuous or on movement).	Tremor: Fine. Coarse.	
		Certain regular movements due to interossei, pronators, etc.	
	Irregular (continuous or on movement).	Choreoid.	Continuous spasms, or inco-ordination of movement.
		Jerking.	
Slow, mobile spasms, of remitting type.	Continuous.—"Athetosis."		
	On movement.—Slow, cramp-like inco-ordination.		"Spastic contracture" of hemiplegic children.
Tonic spasms, varying	Of interossei, conspicuous.		
Fixed rigidity, unvarying.	Of flexor longus digitorum, Conspicuous—late rigidity.		

The following are the clinical facts which seem to call for special attention :

(1.) All these forms of spasm occur almost invariably in cases where voluntary power over the affected muscles is present to a greater or less degree, for example, generally during the recovery from hemiplegia.

(2.) In the vast majority of cases, the movements are unilateral, but occasionally, as in chorea, they are bilateral.

(3.) As a rule, the movements succeed hemiplegic attacks, or other signs of localized cerebral disease, but in some cases they come on gradually, without preceding paralysis.

(4.) The subjects of the disordered movements are sometimes epileptic, and at the time of the seizure the muscles which are the seat of the mobile spasm are first and pre-eminently attacked ; furthermore, the position given to the limb at the outset of the seizure is sometimes that which it is apt to assume under the influence of the semitonic spasms ; again, this same position is sometimes assumed as an "associated movement," whenever the opposite healthy limb is moved.

(5.) Voluntary innervation, even if finally relatively successful in its aim, has usually at first the effect of increasing any existing spasmodic action of the muscles.

(6.) The spasms which are especially the subject of this paper, whether mobile or spastic, affect by preference the smaller, more rapidly acting muscles of the limb (in the upper extremity, the interossei), while the fixed spasms, constituting the so-called "late rigidity" of hemiplegia, affect especially the larger, more slowly acting muscles (long flexors). The slow, mobile spasm (athetosis) is usually confined to the extremities, but occasionally the muscles of the neck and face are affected as well.

The relationship between these different forms is shown by the fact that not only do they originate under similar circumstances (after hemiplegia, etc.), but they are interchangeable in the same person, or the arm may be affected in one way, and the leg, at the same time, in another—thus, in one case, the leg by a mobile spasm and the arm by a fixed spasm. This same interchangeability of the different forms of spasm is shown strikingly in an interesting case, reported by *Kahler* and *Pick*, to which reference will again be made further on. Here, both fingers and toes were in the first place in rapid, ceaseless motion, of the "piano-forte player" type (*Remak*). Later, the movements were more extended and jerky, like those which have been considered as typical of hemichorea; while finally they had the slow character distinctive of athetosis (*Goldstein*).

As will be seen, *Gowers* ranks even the "late rigidity" of hemiplegia among these spasms. At any rate, as he says, it does not deserve to be classed as a permanent contracture, since it varies so much under different circumstances. Contrary to the mobile spasms, it affects the long digital flexors, and respects the interossei.

Hughlings Jackson's theory as to the "late rigidity," that it is of cerebellar origin, is well known, and deserves consideration here.

Oulmont gives an analysis of all the cases hitherto published which seem to him to deserve the name of athetosis, and confirms *Charcot's* opinion that hemianæsthesia is usually present at one time or another in the course of the affection.

Out of twenty-six cases it was noted eleven times, and in a number of others no search had been made for it. In some of these cases, its absence was distinctly recorded, however, as *Gowers* also points out. In all but four of the twenty-seven cases, there had been hemiplegia, and even in two of these four a cerebral lesion of some kind.

Oulmont describes also, under the name of double (bilateral) athetosis, a kindred malady which a few observers have spoken of, but which, on the whole, has been slighted, or perhaps classed as chronic chorea. *Dreschfeld* reports two typical cases, observed at the Idiot Asylum in Lancaster. *Claye Shaw* has described others, and in fact they are perhaps not very rare. I have myself followed two such cases carefully for many years, and have seen, but not studied, a third.

The symptoms, as *Oulmont* shows, date from early childhood, or may even be congenital. They are not preceded by paralysis, but are apt to be associated with idiocy or imbecility.

This was not true, however, of a case seen by *Gowers*, nor of either of the three instances which have come to my own knowledge. The movements are less violent than in those of the typical athetosis, especially so long as the patients are at rest; other muscles of the face are usually involved, as well as those of the limbs. The sensibility is unaffected.

These cases show no tendency to spontaneous recovery, though the patients are capable of increasing their voluntary control very materially by persistent and systematic exercise.

The *prognosis* for the unilateral athetosis is likewise, as is well known, of the poorest; nevertheless, occasionally a case does unexpectedly well. Thus *Gowers* reports one case which recovered entirely under the influence of the galvanic current applied daily, from the back of the neck to the affected muscles; and *Gnauck* another which likewise recovered under the same treatment, with the addition of potassium bromide.

One of these cases was of the post-paralytic variety; the other was idiopathic (or primary), and the symptoms had already been present once, and had disappeared under the use of the bromide of potassium.

PATHOLOGY.

The cases are now sufficiently numerous in which definite anatomical lesions have been found, in one or another of these forms of cerebral spasm, but a pathogenetic theory is still to be expressed only in the vaguest terms, nor is it yet certain how far the different varieties alluded to can profitably be classed together.

Gnauck points out, in a carefully written paper, that the cases of primary athetosis, those namely which are not associated with other symptoms of organic cerebral disease, and which may be either unilateral or bilateral, should be taken as the types of the affection, the remainder constituting the vast majority being classed as "symptomatic."

Gnauck quotes an autopsy recorded by *Oulmont*, the only one which has been made in a primary case of this kind, in which a focus of softening was found in the left corpus striatum and nucleus lenticularis, the symptoms having been confined to the right side.

Until we understand better the physiological pathology of the affection, it seems to me that this distinction of primary and symptomatic must be received with caution.

Kahler and *Pick*, who review the pathological anatomy of the reported cases with great care, consider that the other symptoms of organic disease—the hemiplegia and the hemianæsthesia—are, so to speak, accessory symptoms, the essential pathological condition consisting in a state of irritation of the pyramid-tract (*Flecksig*) in the internal capsule, due to lesions which may or may not be sufficiently severe to cause paralysis and secondary degeneration of the cord.

Twelve autopsies are cited (by *Lépine*, *Raymond*, *Gowers*, *Lauenstein*, *Landouzy*), in nine of which the thalamus opticus was found to be the part mainly involved, in two the nucleus lenticularis, and in three the internal capsule, all these regions bordering immediately on that portion of the internal capsule which is traversed by the continuation of the pyramid-tracts. To these is to be added the instructive case reported by themselves, where the outer half of the thalamus opticus was diseased, and at one point, opposite the posterior end of the nucleus lenticularis, the internal capsule in its whole thickness.

As additional evidence that an irritative process in the great motor tracts is the cause of these symptoms, a case is quoted from *Ewald*, where analogous movements were found to have been probably due to the presence of a small tumor in the right anterior half of the pons, in close contact with the pyramid-tract; also, from the *Lancet* of 1871 (p. 852), another case, of crossed paralysis of the face and limbs, with mobile spasm of one arm and choreic movements of both legs, due presumably to glioma in the fourth ventricle.

Another instance of a like kind, though without autopsy, is given by *Gowers*, and one is also quoted by him from *Bastian*, where the symptoms indicated lesion of the crus cerebri. The bilateral occurrence of the symptoms in some cases may perhaps, likewise, point to the pons as the seat of the lesion.

The observation of *Rosenbach*, now confirmed by every one, that patients with locomotor ataxia often have slight, involuntary, slow movements of the hands and feet, is also brought up to show that these symptoms of mobile spasm may be associated with lesions low down in the cord. Much physiological work remains to be done, however, before we can claim to have an accurate conception of the pathogenesis of this class of symptoms.

It is not to be forgotten that *Ewald* and *Kuessner* report cases of athetoid movements of typical character, associated, in the one case with localized cortical lesions (temporal lobe), in the other with no macroscopic lesion whatever. The patients were, however, in both cases general paralytics. The hemianæsthesia in these cases of athetosis is, of course, readily explained by the nearness of the lesion to the posterior region of the internal capsule, and I cannot but think, with *Charcot*, that the essential lesion for many cases will turn out to be in the centripetal rather than in the centrifugal tracts.

A very interesting case was recorded by *Ringer* in 1877, of athetosis with temporary hemiplegia and hemianæsthesia, and accompanied by unilateral sweating, all on the right side. Within a year an account of the autopsy has been published, which shows that there was very extensive disease of the left corpus striatum, nucleus lenticularis, and thalamus opticus.

Ringer holds the view which, if it be expressed sufficiently vaguely, is essentially like that held by *Gowers*, *Jewell*, *Kahler* and *Pick*, and no doubt others, that the symptoms are due to the spreading (irradiation) of the motor impulses passing from the cortex cerebri to the muscles (or coming as centripetal excitations from other parts of the nervous system) within these damaged tracts, so that they set up what, in a physiological sense, might be called associated movements. Furthermore, it is supposed that, in virtue of the disordered nutrition within the masses of affected gray matter spontaneous motor impulses may originate in them.

It is certainly unscientific to speak of these lesions, as so many do, as causing the characteristic movements by direct irritation of motor tracts.

When one reflects on the character and regularity of the movements and their persistency, it is far more philosophical to regard them as the effects of the activity of one or many organized nervous centres, and to speak of them rather as "permitted" by the removal of inhibitory control than as due to irritation.

Any comprehensive theory would have to take into account the fact that these mobile spasms are sometimes associated with returning control after hemiplegia, sometimes are entirely independent of hemiplegia, and occasionally (*Bernhardt*) occur in company with complete paralysis.

With regard to *treatment*, the favorable action of galvanism and potassium bromide in a few cases has been referred to above.

CHOREA.

BIBLIOGRAPHY.

de Boyer, H. C.: Chorée cardiaque. Progrès méd., 1875, No. 52.—Dickinson, H.: Pathology of Chorea. Lancet, 1875, II., p. 559.—Rosenbach, O.: Zur Pathologie und Therapie der Chorea. Archiv für Psychiatrie und Nervenkrankheiten, Bd. VI., S. 830, 1876.—Heller, Fr.: Angeborene Chorea. Wiener med. Wochenschrift, 1876, Nr. 19.—Simpson, A. R.: Fatal case of Chorea Gravidarum. Obstetric. Journal, XXXVII., 1876, p. 80.—Sturges, O.: Chorea and Whooping-cough. Five lectures. 16mo, London, 1877.—Spamer, C.: Zur Casuistik der Chorea (Congen.). Wien. med. W.schr., 1876, No. 52 (Cbl. für med. Wissen.), 1877, p. 336.—Rocher: Note sur un cas de chorée dans la vieillesse. Bull. Soc. de méd. du dep. de la Sarthe, 1877, Le Mans, 1878, 32-38.—Luton, A.: Existe-t-il une névrose qui mérite specialement le nom de chorée rheumatismale? Mouvement méd., Par., 1877, XV., 364-366.—Charcot: A Lecture on rhythmical Hysteric Chorea. Brit. M. J., Lond., 1878, I., 224, 251.—Bourneville: De la chorée vulgaire chez les vieillards. Progrès méd., Par., 1878, VI., 177.—Gowers, W. R.: On some Points in the Clinical History of Chorea. Brit. Med. J., Mar. 30th, 1878.—Charcôt: On Chorea in old People. Med. T. and Gaz., March 9th, 1878.—Berdinet, P.: Chorée des adultes. Gaz. méd. de Paris, No. 28, 1878.—Russell, J.: Note on a Case of Chorea in an aged Person, followed by Recovery. Med. Times and Gaz., Lond., 1878, I., 459-460.—Gee: Chorea with Pericarditis and Endocarditis. M. T. and Gaz., Lond., 1878, I., 506. —Takács, E.: Chorea electrica (Case) Orvosi het. Budapest, 1878, 416.—Peacock, T. B.: Statistical Report on Cases of Chorea. St. Thomas Hosp. Rep., Lond., 1878, N. S., VIII., 1-30.—Putzel, L.: Cerebral Complications of Chorea. Med. Rec., N. Y., 1879, XVI., 220-222.—Schreiber: Ein Beitrag zur Chorea Laryngis. Wien. med. Bl., 1879, II., 350.—Gowers, W. R., and Sankey, H. R. O.: The Pathological Anatomy of Canine Chorea. Med. Chir. Tr., Lond., 1877, LX., 229-247, 2 pl. Also Proc. Roy. M. and Chir. Soc., Lond., 1877, VIII., 214-216.—Seifert, O.: Beitrag zur Pathologie und Therapie der Chorea minor. Aus der Erlanger medicinischen Klinik. Deutsches Arch. f. klin. Med., Leipz, 1877, XX., 319-335.—Schultze, F.: Zur pathologischen Anatomie der Chorea minor, des Tetanus und der Lyssa. Deutsches Arch. f. klin. Med., Leipz., 1877, XX., 383-396, 1 pl.—Sturges, O.: Lectures on the Clinical Aspects of Chorea, as affording a Key to its Pathology. Med. Times and Gaz., Lond., 1877, I., 357, 437, 607, 635.—Arlidge, J. T.: The Pathology of Chorea. Brit. M. J., Lond., 1877, VII., 799-800. —Hutchinson, J. H.: A fatal Case of Chorea. Tr. Path. Soc., Phila., 1877, VI., 130–139.—Allison, R. H.: Some Cases of syphilitic Chorea. Am. J. Med. Sc., Phila., 1877, II., 75–81.—Dowse, T. S.: The Embolic Theory of Chorea. Brit. M. J., Lond., 1877, I., 38–39.—Bastian, H. C.: Remarks on the Pathology of Chorea. Brit. M. J., Lond., 1877, I., 36, 65.—Kretschy, F.: Ein Fall von Chorea mit lethalem Ausgange. Wien. med. Bl., 1878, I., 329–331.—Simpson, A. R.: Note of a fatal Case of Chorea Gravidarum. Tr. Edinb. Obst. Soc., 1878, IV., 238–241.—Frank, E.: Ein letal abgelaufner Fall von Chorea minor. Allg. Wien. med. Ztg., 1879, XXIV., 367.—Putnam, J. J.: Two Cases of Chorea in the Kitten, with Autopsies. Boston M. and S. J., 1879, CI., 690–692.—Weiss, Nathan: Ueb. die therapeutische Verwendung des Propylamin in einigen Nervenkrankheiten. Wien. med. Blätter, 8 S., 186, 9 S., 206. —Tidemand: Norsk. Magaz. for Lægerid, R. 3, Bd. 8. Treatment of Chorea by Gymnastics.—Wood, H. C.: Chorea, its Etiology and Varieties, with a new Treatment; a clinical lecture. Med. Record, N. Y., 1877, XII., 785–786.—Barclay: A case of Chorea with Typhoid Fever supervening. Med. Examiner, Lond., 1877, II., 511–512.—Bernheim, A.: Observation de chorée générale compliquée de fièvre typhoide. Mém. Soc. de Méd. de Nancy, 1877, 53–55.—Dickinson, W. H.: On the Treatment of Chorea. Lancet, Lond., 1877, I., 3–5.—Drummond, D.: A Case of Chorea treated by the Subcutaneous Injections of Curara. Brit. M. J., Lond., 1878, I., 857.—Purckhauer, H.: Propylamin gegen Chorea minor. Aerztl. Intell. Bl., München, 1878, XXV., 1–4.—Richter, F.: Zur Therapie der Chorea minor. Deutsches Arch. f. klin. Med., Leipz., 1878, XXI., 373–387.—Stone, W. H.: Thymol in Chorea. Med. Times and Gaz., Lond., 1878, I., 261–262.—Gray, L. C.: The Arsenical Treatment of Chorea. Proc. M. Soc. County Kings, Brooklyn, 1878, III., 273–298.—Sturges, O.: On the Management of Chorea. Med. Times and Gaz., Lond., 1878, I., 302, 559; II., 69–70.—Van Bibber, J.: The new Treatment of Chorea, 8vo, Baltimore, 1878 [Reprint from Trans. M. and S. Faculty of Maryland, 1878].—Wright, R. T.: Subcutaneous Injection of Curara for Chorea. Lancet, Lond., 1878, II., 253.—Hammond, W. A.: On the Treatment of Chorea with hypodermic Injections of Arsenic. St. Louis Clin. Rec., 1879-80, VI., 193–196.—Garin, Henri: Du traitement de la chorée spécialement par l'arsenic et les injections hypodermiques de liquor de Fowler, Lyon, 1879, 90 pp. 4to, S. No. 14.—Dresch: Chorée guérie en huit jours à la suite de l'emploi du salicylate de soude. Bull. gén. de thérap., etc., Par., 1879, XCVI., 506.—Mitchell, S. W.: Chorea and its Treatment by Salicylate of Soda. Boston M. and S. J., 1879, C., 447.—Sturges, O.: The Pathologies of Chorea in their Application. Med. T. and Gaz., Lond., 1880, I., 257, 286.

ETIOLOGY AND SYMPTOMATOLOGY.

An interesting illustration of the suddenness with which fright may bring on chorea is given by *Henry Day*, who was present at the very moment of the attack. A passer-by, on a country road, saw a boy (nine years old) climbing an apple-tree, and, thinking him to be in a dangerous position, indiscreetly called to him to come down. The boy, alarmed, lost his hold and fell to the ground, striking on the front of the body. He had received no injury, but, as soon as he had recovered his breath, shouted and screamed with seeming fright, and "then and there" the symptoms of bilateral chorea showed themselves, first in the arms and shortly afterwards in the legs. He recovered in less than a month.

The opinion advanced in the early edition of this Cyclopædia, that an intimate relation may exist between chorea and rheumatism, is still upheld by most writers, though as to the nature of this connection we are fully in the dark. *v. Ziemssen* (Cyclop., 2d Ed.) reports at length an interesting case where chorea, in a violent form, developed during the course of an acute articular rheumatism, with endocarditis and pleuritis, and disappeared again in the course of three weeks, even before the articular symptoms had entirely gone. *Gee* gives a similar case, where, however, the movements were slight in amount, and the pains in the joints slight. There was peri- and endocarditis, with a fatal issue. Nothing whatever could be found in the brain to account for the choreic symptoms. Endocarditis may occur in association with chorea without, so far as is known, having been due to rheumatism. This is illustrated by two cases reported by *Boyer* (v. 2d Ed. *v. Ziemssen*, p. 465) in addition to those recorded in the first edition of the Cyclopædia.

Finally, *Mitchell* and others have noted cases in which rheumatism has immediately followed chorea, instead of preceding it.

With regard to the season of the year in which chorea is the most prevalent, *Gerhard* gives an analysis of eighty cases coming under his observation in Philadelphia which seems fully to confirm the view of Dr. *Mitchell* and himself, that by far the greater number of cases, and especially relapses, begin in the spring. In recent publications *Mitchell* still adheres to this opinion.

In the experience of others, this fact has not been fully confirmed. An analysis of the cases which have come under my own observation, in the Out-Patient Dep. of the Mass. Gen. Hosp. during the past nine years, would show that the winter and spring are in Boston about equally prolific, the six months from December to June bringing in a much larger number of cases than the other half-year. In other climates perhaps another rule would prevail.

To the cases mentioned by *v. Ziemssen* of chorea occurring in aged people may be added a few isolated instances (*Russell, Berdinet*).

Charcot contributes a clinical lecture on this subject, in which he says, and *Berdinet* essentially agrees with him, that this chorea is often associated with dementia, and not especially connected with acute articular rheumatism, or even with rheumatic gout, and that it is incurable.

Gowers calls attention to an important point which has never been dwelt upon in the descriptions of the choreic motor symptoms, namely, that cases differ greatly in respect to the proportion which the involuntary movements bear to the loss of voluntary co-ordinating power. *Gowers* believes he has noticed that "when such a disproportion exists, the inco-ordination is in excess of the spontaneous spasm early in an attack, and the spontaneous spasm is in excess of the inco-ordination late in an attack and during relapses."

Sturges also calls attention to an (apparent) lack of co-ordinating power, in an interesting series of papers, to be referred to later, but thinks it is not a true ataxia, in the sense of ataxia from organic disease, inasmuch as it may be now present, now absent, or, at any rate, vary greatly in amount at different times.

Gowers further observes that:

1. The choreic movements may be confined to one arm, both legs being free from movement, although that in the arm is violent.

2. When the chorea affects considerably one arm and leg, although the other arm is quite free from movement, it will generally be found, on close examination, that there is some degree of affection of the other leg.

3. Occasionally the affection of the other leg is as great as that of the leg of the side on which the arm is affected.

"The affection may thus be unilateral in the arms, bilateral in the legs." If the observation is confirmed, it will be one, as is held by *Gowers*, to place parallel with the physiological fact that the use of the legs is predominantly bilateral, that of the arms unilateral. *Gowers* has observed in several cases, in confirmation of the statements by *Rosenthal* and *Benedikt*, that the irritability of the nerves of the affected limbs, in hemichorea, is greater than that of the nerves of the opposite limbs.

He could not satisfy himself, however, that the opening kathode-reaction was greater than normal in the same cases.

PATHOLOGICAL ANATOMY.

There is but little that is important to record on this point. *v. Ziemssen's* opinion still inclines toward the embolic theory, first advanced by *Jackson*, on interesting theoretical grounds. Besides two cases reported by *Jackson* where emboli were found, the latter quotes *Bastian* as having discovered them in three others. *Dickinson* examined seven cases without succeeding in finding them, but observed, instead, intense congestion of the cord, with occasionally small hæmorrhages, and marked congestion also in various parts of the brain, especially the corpora striata.

Congestion also was the marked feature in the cases of *Hutchinson* and *Kretschy:* in the former's case there was also thickening of the cerebral arachnoid here and there, and localized softening in the cord. In *Gee's* case, though peri- and endocarditis were present, nothing of great importance could be found in the brain.

Gowers and *Sankey* have reported two examples of chorea in the dog, with careful examination of the nervous centres. In one case the movements were universal and so severe that, through them and some paresis of the hind legs the animal was unable to walk, though he could move his limbs and tail with a good deal of force while lying on the ground. After the cervical cord was divided, the movements ceased in the parts supplied by the spinal cord, but continued in those supplied by the medulla oblongata. On examination, there was found scattered irregularly through the cord, medulla, and cerebellum, but not in the hemispheres or corpora striata, a marked leucocytal infiltration of the tissues adjacent to the blood-vessels, in both the white and the gray substance, and likewise a granular, slightly disintegrated condition of the nerve-cells. In the second case, in which the movements had been slighter and confined to one fore-leg, none of this vascular change, except "turgescence" here and there, was found, but the nerve-cells in the right half of the cervical enlargement in its lower half (whether on the same side with the affected limb was not known) were found swollen and granular. A similar change was present in the right posterior vesicular column in the lumbar region. The authors regard the change in the nerve-cells as secondary to a functional "over-action," and the vascular condition as a further result. I have recently had the opportunity of examining the brain and cord of a kitten affected with the disease in an absolutely typical form—one of four out of a large and healthy litter that had presented the same symptoms—and could discover absolutely no deviation from the normal state. In the second case of *Gowers* and *Sankey*, the movements persisted after section of the spinal cord in the cervical region.

On the whole, it must be confessed that anatomical study lends but little support to the theory which supposes chorea to be of embolic origin, or of definite organic origin at all. The objections to these theories are well summed up by *Sturges*, who looks upon the affection as of functional origin and analogous to hysteria. His arguments are forcible, and deserve careful consideration, especially as they bear directly upon the question of treatment.

TREATMENT.

The arsenical treatment of chorea still counts the warmest adherents, in spite of newer remedies, and further evidence is furnished that, if given by subcutaneous injection, the effects are better than when it is used by the stomach, and the chance of gastritis diminished. *Garin* has had excellent results in thirty-three cases, giving in this way four to five drops of Fowler's solution, properly diluted, every few days. *Hammond* goes up to much higher doses than these, even ten drops to a child of eight years, and thirty-five drops to an adult. He recommends the neighborhood of the deltoid as the best place to inject.

Sometimes, as *v. Ziemssen* points out, the injections cause pain and inflammation, and it is to be remembered that the fatty degeneration of the glands of the stomach is in part an effect of the constitutional as well as the local action of the poison, and is, therefore, liable to occur even when it is given hypodermically.

Weir Mitchell has treated a number of cases, with excellent results, by sodium salicylate, tried first in cases complicated with rheumatism, but found efficacious as well in others. *Dresch* has had equal success with this treatment in one case.

Propylamine, in dose of 1.0 to 1.50 *pro die*, divided into hourly doses, has been successfully employed (*Purckhauer*, *Nathan Weiss*, quoted in *Virchow* and *Hirsch's J.bericht*), and *Drummond* and *Wright* speak favorably of subcutaneous injections of curare. I have had but little experience with either of these remedies, and no general judgment has been passed on them.

The books often speak of the favorable action of gymnastics in chorea, but it is perhaps rarely appreciated that, to get these results, a degree of persistence and system is usually required which implies trained supervision and the expenditure of much time. This is illustrated by the description of a method successfully used by *Tidemand*, in a case of a year's standing (*Virchow* and *Hirsch's J.bericht*), where the patient was kept on his back most of the time for two or three months, and was exercised daily by a skilled teacher once every twenty or thirty minutes through four hours in the forenoon and three in the afternoon.

In judicious hands, some sort of moral management, as is urged by *Sturges* as a substitute for all medicinal treatment, might be eminently useful. It is founded on his belief that the symptoms of the disease are closely associated with the operation of the higher cerebral functions in their relation to the muscular system, and aims at "diverting the child's attention from its perverted movements, while at the same time tacitly discountenancing them."

HYSTERIA.

BIBLIOGRAPHY.

Lotz: Die psychische Lähmung d. Stimmbänder. Correspondenzbl. f. schweizer Aerzte, 1873, Nr. 16.—Schnitzler: Ueb. Sensibilitätsneurosen d. Kehlkopfs. Wien. med. Presse, 1873, Nr. 46.—Svynos: Des amblyopies et des amauroses hystériques. Thèse de Paris, 1873.—Cartiaux: Hystérie confirmée chez une femme privée de vagin et d'utérus. Gaz. des hôp., 1873.—Fernet: De l'oligurie et de l'anurie hystériques et des vomissements qui les accompagnent. Union méd., 1873.—Bourneville: Etudes cliniques et thermométriques sur les malad. du syst. nerveux. 2. fascic., 1873.—Penzoldt: Ueb. d. Paralyse der Glottiserweiterer. Deutsches Archiv f. klin. Med., Bd. XIII., 1874.—Emminghaus: Wirkung der Galvanisation am Kopfe bei Aphonie. Archiv für Psych. und Nervenkrankheiten, Bd. IV., 1874.—Ferran: Du vomissement de sang dans l'hystérie. Paris, 1874.—Lorey: Des vomissements de sang supplémentaires des règles. Thèse de Paris, 1875.—Fabre: De l'hystérie chez l'homme. Annal. méd. psych., 1875.—Bonnemaison: Sur en cas d'hystérie chez l'homme. Arch. génér. de méd., 1875.—Bonnefoy: Des troubles de la vision dans l'hystérie. Thèse de Paris, 1874.—Bach: De la coxalgie hystérique. Thèse de Paris, 1874.—Lafou: De la toux hystérique. Thèse de Paris, 1874.—Hudson: Epidemic of Hysterical Epilepsy and Tetanus. Lancet, 1875.—Holst: Ueb. Neurasthenie und ihr Verhältniss zur Hysterie und Anämie. Dorpater med. Zeitschr., Bd. VI., Hft. 1, 1875.—Da Costa: Hysteria with Lung Symptoms Simulating those of Phthisis. Phila. Med. T., 1878, VIII., 267.—Lasègue, C.: Des hyst. périphériques. Arch. gén. de méd., 1878, CXLI., 641.—Fürstner: Ueber die Anwendung des Inductionsstroms bei gewissen Formen der Magenerweiterung. Berl. klin. Wochenschr., 1876, Nr. 11.—Tittel: Ein Fall von Hämathidrosis. Arch. f. Heilkunde, XVII. Heft 1, 1876. —Seeligmüller, A.: Ueber epidemisches Auftreten von hysterischen Zuständen. Allg. Zeits. f. Psychiatrie, Berlin, 1877, XXXIII., 510–528.—Reynard and Richer: Etudes sur l'attaque hystéro-épileptique faites à l'aide de la méthode graphique. Rev. mens. de méd. et de chir., Par., 1878, II., 641–661.—Charcot: On Disturbances of Sight in Hysteria. Med. Times and Gaz., Lond., 1878, I., 55–56.—Charcot: Episodes nouveaux de l'hystéro-épilepsie. Zoopsie. Catalepsie chez les animaux. Gaz. d. hôp., Par., 1878, LI., 1097–1099.—Charcot: L'attaque hystério-épileptique. Gaz. d. hôp., Par., 1878, LI., 1121–1123.—Charcot: Details complementaires relatifs à l'hystéro-épilepsie. Gaz. d. hôp., Par., 1878, LI., 1145-1147. Potain: Hystérie à forme vésicale. Gaz. d. hôp., Paris, 1878, LI., 281-282.—von Hesse: Hemianæsthesia hysterica. Arch. f. Psychiat., Berl., 1879, X., 271-273. —Scholz, G.: Ueber Hystero-Epilepsie. Arch. f. Psychiat., Berl., 1879, IX., 636-662.—Buzzard, T.: Hysterical Hemianæsthesia of the Left Side, Defective Electrical Excitability of the Right Cerebral Hemisphere. Lancet, Lond., 1879, II., 685.—Roberts, W.: Cases of Hysteria in Boys. Practitioner, Lond., 1879, XXIII., 339-345.—Welponer: Eine Frau, an welcher wegen Hystero-epilepsie die Entfernung beider Eierstöcke vorgenommen wurde. Anz. d. k. k. Gesellsch. d. Aerzte in Wien, 1879, 148.—Poirier, P.: Des moyens d'arrêter les attaques hystéro-épileptiques et en particulier du compresseur des ovaires. Progrès méd., Par., 1878, No. 52, VI., 993.—Le Rolland, Joseph: Considérations sur l'influence de la grossesse sur la marche de l'hystérie et de l'épilepsie. Par., 1879, 56 pp. 4to, No. 92.—Shaffer, N. M.: The Hysterical Element in Orthopædic Surgery. Arch. of Med., N. Y., 1880, III., 40-56.—Schmidt: Ueb. das Vorkommen der Hysteria bei Kindern (Inaug. Diss.). Strassburg, 1880, Cbl. f. N. Heilkunde, etc., 1880, No. 9 (Schaefer)

Metallo-Therapeutics, Etc.

Charcot: Lond. Lancet, 1878, No. 3 et seq.—Charcot, Luys, Dumontpallier: Brit. M. J., 1878, II., 548. Many papers in the Gaz. hebdom., and Progr. méd. for 1877, 1878, 1879.—Bourneville, Regnaud, Vigouroux, Raynaud, and others vide gen. index art. "Met. Ther.," also under Soc. de Biol.).—Westphal, Bernhardt: Ber. klin. Wochenschr., 1878, July 29th and March 11th.—Wilks: Brit. Med. Jr., 1878, July 20th.—Eulenburg: Deutsche med. Wochenschrift, 1878, Nos. 25 and 26.—Bennett: Brit. Med. Jr., Nov. 23d, 1878.—Tuke: Jr. Ment. Sc., Lond., 1879, N. S., XXIV., 598.—Vierordt: C.bl. f. d. med. Wissensch., 1878, XVII., 1. —Sigerson: Brit. M. J., Lond., 1879, I., 143.—Beard: Pop. Sc. Monthly, May, June, July, 1878. Chicago Jr. of Nerv. and Ment. Dis., 1877 and 1878. A Practical Treat. on Nervous Exhaustion, 1880, N. Y.—Petit: Bull. gén. de thérap., 1879-1880.—Garel: Lyon méd., 1880, XXXIII., 53.—Franz Müller: C.bl. f. Nervenheilkunde, etc., 1879, 26.—Erlenmeyer: C.bl. f. Nervenheilk., etc., 1879, 315.—Bennett: Brain, 1878, III., 331.

ETIOLOGY.

In the second edition of the Cyclopædia, *Jolly* quotes *Parant-Duchatelet* (La prostitution dans la ville de Paris) as saying that prostitutes suffer relatively seldom from hysteria, but ranges himself with *Scanzoni* as of the opposite opinion.

The etiological moment is not purely, however, in their case, the excessive genital irritation, but in part the excitement and unfavorable hygienic conditions amidst which this class of women live.

Jolly speaks also of the fact that bodily injuries, such as falls, blows on the head, and the concussion of railway accidents, may act as an exciting, and possibly also as a final cause of a condition to which the name hysteria could hardly be denied; though, in the case of the railway accidents at least, it is probable that the mental excitement and agitation play at least as important a part as the concussion.

Rustic populations afford a less number of the lesser hysterical affections—those which border on neurasthenia—than the civic. Still, as *Jolly* justly remarks, the severest examples of true hysteria often come from the country. This is well illustrated by an interesting case reported by *Gairdner*, occurring in a girl of eighteen, of cheerful and even lively disposition, living happily in strict retirement, with two aunts, visiting and visited by nobody, of fair general health.

SYMPTOMATOLOGY.

The systematic study of hysteria, in all its branches, has been vigorously pursued in France during the past few years, especially by *Charcot* and his pupils, in the great field of La Salpêtrière at Paris, and it is, therefore, to the results of their labors that I shall principally refer, besides citing briefly the most important facts added by *Jolly* in the second edition of the Cyclopædia.

Charcot and *Galezowski* have investigated with care the hysterical loss of vision, and find that color-perception of these patients disappears according to a certain law, violet being the first color to go, and blue the last, for most cases, though it occasionally happens that the perception for red is the last to disappear, these cases forming then another type. The usual order is violet, green, red, orange, yellow, blue. These observations are important, because it would appear from them that the perception for the different colors is lost in hysteria in the same order as from causes of different nature.

A case of hysterical amaurosis, of great persistence, is reported by *Mendel* (*Jolly*). It came on suddenly, not after an attack, lasted eight months, and then disappeared entirely. The results of the ophthalmoscopic examination were invariably negative.

Cases of ataxia in hysterical patients have occasionally been observed, the inco-ordination sometimes affecting single muscles (especially those of the eye), sometimes an entire limb, sometimes the entire muscular system, coming from time to time and disappearing again spontaneously. Two cases, possibly belonging in this category, have been under my own care, where there was room for doubt whether the true diagnosis was hysteria or disseminated sclerosis. Both the patients were highly nervous, unmarried women; the inco-ordination was confined to one hand, appeared only during voluntary efforts, and was of a year or more's duration at the time the patients first presented themselves.

Cases of paralysis of the abductors of the vocal cords are reported by *Penzoldt*, *v. Ziemssen*, *Johnson*.

Lasègue has described, under the name of "hystérie péripherique," a group of cases of considerable interest, characterized by the fact that, although the patients do not necessarily exhibit the general hysterical temperament, the slightest peripheral irritation causes in them muscular spasms, which persist with extraordinary obstinacy, finally perhaps to disappear with great suddenness. Such are certain cases of rheumatic torticollis, and of blepharospasm from slight and passing irritation of the conjunctiva. *Lasègue* says: "The transition from the typical hysteria to the other (functional) diseases of the nervous system is not abrupt, but by imperceptible gradations. The term "hysteroid" as well deserves a place in our nomenclature as 'epileptoid,' 'rheumatoid,' etc."

Roberts and *Schmidt* bring additional evidence of the occurrence of hysteria in children.

The differential diagnosis between the contracture of hysteria and that due to joint-disease has been carefully studied by *Shaffer*. Both forms yield to the complete anæsthesia of ether and chloroform, but the former alone is said to disappear during sound sleep, whether natural, or induced by moderate doses of chloral and opium.

In hysterical contracture, moreover, there is only the atrophy of disuse, with but little or no change in faradic reaction; in contracture from joint-disease, the atrophy is more marked, and the faradic reaction diminished.

The phenomena constituting the attacks of hystero-epilepsy, or hysteria major (*Charcot*) have been investigated with the utmost care by *Charcot* and his pupils at the Salpêtrière, even to the extent of registering the different phases of the muscular spasm by means of Marcy's tambour ingeniously attached to the forearm.

The seizure as a whole is regarded as a phenomenon of pure hysteria at bottom, the epileptic element affecting solely the external form of the paroxysm.

The attack is said to be preceded by prolonged auras of various kinds, epigastric, cardiac, auditory, visual, etc. Then occurs, first, the epileptoid seizure, with its tonic and clonic phase, the whole lasting, together with the period of resolution which forms the longest part, four to five minutes. The muscular spasms of this stage are cut short, in Paris, both by compression over the ovaries and in other ways.

Poirier has even devised a mechanical contrivance to be worn constantly, with pads resting in the ovarian region, which could be rapidly pressed inward at the outbreak of the attack.

On the other hand, *Gowers* has not been led to attach the same importance with the French writers to ovarian compression, as a means either of provoking or arresting these attacks.

The first period then gives place to the second, that of the contortions, which consist in co-ordinated convulsions of the most varied kind throwing the body into every conceivable attitude.

The third stage can best be imagined if the patient be supposed to be passing through a series of exciting, emotional dreams, gay, sad, almost always obscene in character, and illustrating his mental states by extravagant but fitting gestures and attitudes.

In the fourth, the terminal stage, the patient's mind is filled with terrifying visions, often representing animals of various and unusual kinds, which, *Charcot* says, are apt to appear and disappear towards the anæsthetic side of the body, if hemianæsthesia is present.

The occurrence of such definite attacks as these has not been signalized by writers in other countries (compare, for example, the descriptions given by *Gowers* in his recent lectures on epilepsy), perhaps, because of the fact, which is beyond question, that the characters of hysterical affections differ greatly among different races of people, as well as at different times and places.

Thus *Brodie* long ago pointed out the greater frequency with which hysterical joint diseases are met with in England than in other lands.

PATHOLOGICAL ANATOMY.

In the second edition of this Cyclopædia *Jolly* reviews the differing theories which have been proposed, to explain, or more closely define, the pathological condition of the nervous system which underlies hysteria, without, however, giving in his adherence to either of them. Thus, impoverishment or abnormal composition of the blood is often absent. Erethism (reizbare Schwäche) or irritability with exhaustibility, is not always to be made out. The attempts of recent writers to elevate the latter condition into a clinical entity under the name of neurasthenia are characterized as artificial and unpractical, and it is claimed that almost all the cases on which the symptom-group is based belong either to hysteria or to hypochondria, or else are instances of the effect of outside influences of various kinds upon a nervous system naturally feeble. Since *Jolly* wrote, *Beard* has published a comprehensive work on this subject, in which references are given to the literature of the matter and a careful attempt is made to establish the symptomatology of neurasthenia on a firm basis. To this the reader is referred.

My own view of the question is that it is useful to classify cases under the headings of neurasthenia, hysteria, and even to make still further subdivisions so fast as a sufficient number of similar cases can be found to justify them, but to do so purely and avowedly for reasons of clinical convenience, recognizing the fact that these different groups shade insensibly into one another, or occur mixed in varying proportions; that many cases present themselves which belong in neither distinctly, but in an intermediate class; and, finally, that we know nothing of the pathology of any of them.

Probably the first real step towards unravelling the mystery of these affections will come from the side of physiology and psychology.

An indication of the direction in which more light is to be sought is perhaps already furnished by the various writings of *Hughlings Jackson* and others, as well as in the definition given by *Jaccoud* that the characteristic of hysteria is a predominance of the involuntary innervation over the voluntary innervation.

That typical cases of neurasthenia are distinguishable from typical cases of hysteria, both as regards their etiology, their clinical history, and the manner in which they respond to treatment, is hardly to be doubted, or to put it better, the symptoms described by *Beard* are so well marked and occur so often in company, as to deserve a separate clinical designation, especially as they are almost as common in males as in females. Typical hysterical symptoms may, however, and often do occur in the course of neurasthenia, the same soil serving for the growth of both affections. Of this, the following case may pass as an illustration.

A young lady, between twenty and thirty, intelligent, of well-balanced temperament, and belonging to a healthy family, was prostrated for two or three years with the typical symptoms of neurasthenia (exhaustibility, tender spine, photophobia, neuralgia, with but few fleeting or emotional symptoms) and gradually recovered to a large extent. One day after this, while feeling pretty well, she received some trifling injury in one hand, of which the local symptoms amounted to little or nothing. Shortly afterwards, however, she began to suffer from a sense of powerlessness, subjective disturbances of sensibility, and some pain in the hand, arm, and eventually leg, trunk, and face, on the side of the injury, without there being any external sign of disease whatever, and these symptoms have persisted for nearly a year, constituting what might be called a hysterical hemi-paræsthesia.

METALLO-THERAPEUTICS.

Any account of the recent investigations on hysteria would be incomplete without some mention of the remarkable experiments of which the great hospital La Salpêtrière has been the chief theatre, and which are indicated in the terms metallo- and magneto-therapeutics, although those names utterly fail to suggest the scope of the present investigations. The main facts are briefly as follows: As long as thirty years ago, Dr. *Burq*, of Paris, insisted upon the effect of the cutaneous application of isolated pieces of metal in curing the anæsthesia of hysterical patients, asserting that the metal found to have this action—for all patients did not react to the same kind—would, if taken internally, have a very beneficial influence on the general condition of the patient. For a long time *Burq's* statements obtained no credence, but at last *Charcot* interested himself in them, and with *Burq* himself and others began to make systematic researches in the wards of the hospital. The usual mode of procedure was to take a patient with hemi-anæsthesia, which was so complete that large pins could be thrust through the most sensitive portion of the skin without causing pain, often involving also the nerves of special sense, and place on the forearm or the leg a few discs of metal, isolated from each other, but fastened on the under side of the same strap.

In fifteen or twenty minutes, the sensitiveness would begin to return to the skin above and near the metallic discs, and then gradually to the whole anæsthetic region, and at the same time the corresponding parts on the opposite side of the body would begin to lose their sensibility, until eventually the anæsthesia had complely transferred itself.

Usually these effects disappeared soon after the removal of the metals. Not only cases of hysterical anæsthesia, but a small number of those where the anæsthesia had been associated with organic cerebral disease, have been thus successfully treated. The therapeutic interest of these discoveries has, however, now become entirely overshadowed by their physiological interest.

It was then found that the electrical currents, so feeble as to be appreciable only to a delicate galvanometer, would exert a similar action with the metals; also that not only could anæsthesia be removed or transferred in the manner stated, but hysterical contractures, of years' duration, could be likewise cured or transferred to the opposite limb, by bringing a large magnet into the neighborhood of the affected part or inclosing it in a solenoid, etc.

Whatever interpretation one may choose to adopt for these phenomena, some of their features will always present a peculiar scientific interest, and this will attach not least to these observations with regard to the transfer of symptoms from one side to the other, as showing a sort of functional antagonism between them, which, perhaps, finds its parallel in the so-called antagonism between the two retinæ. In the recent hypnotic investigations of *Haidenhein*, the independence of the two halves of the body, under certain conditions, is strongly brought out.

The metallo- and magneto-therapeutic experiments of *Charcot* have been repeated the world over, with every possible modification, and have already given birth to a voluminous literature. It would be impossible to follow the subject in all its branchings and not even desirable to do so, since to interpret fairly all the evidence would be a task only suited to an expert, both in physiology and still more in experimentation upon human beings, yet without such interpretation the bare facts would soon lose their interest. I shall, therefore, content myself with mentioning the most significant discoveries. In the first place, it has shown itself clearly that not metals and magnets alone, but various other substances, such as mustard plasters, bits of wood, metals with silk coverings, vibrating rods, etc., are capable of producing the results mentioned, wholly or in part. On the other hand, *Charcot* and *McCall Anderson* claim to have found that, when false magnets were substituted for true ones, or electromagnets deprived of their magnetism without the knowledge of the patients, the results failed to appear. *Schiff* brought about what he believed to be tactile anæsthesia of a dog's fore-paw, by extirpating the corresponding portion of the cortex cerebri in the parietal region, so that cutaneous irritation no longer caused reflex movements as on the healthy side, and found the sensibility return after the limb had been exposed to the action of a solenoid. This important experiment has not, so far as I know, been repeated by others, and all observes do not agree with *Schiff* and *Munk* that cutaneous anæsthesia exists after the lesion alluded to. *Vierordt* tried the experiment of laying pieces of zinc upon the belly of frogs, after first destroying the cerebral hemispheres to prevent voluntary movements, and showed that thereon the reflex contractions which followed stroking and pinching the toes occurred much more readily.

On the other hand, neither *Schiff* nor *Gamgee* was able to excite changes in the physiological properties of the bared sciatic nerve of the frog by exposing it to the influence of strong electro-magnets.

If we regard the clinical facts from the purely physiological side, we are able to find as yet no sufficient cause for the removal of the anæsthesia, and the other phenomena alluded to.

There is no physiological influence common to all the agencies which have produced these effects except possibly a feeble excitation, or, according to *Schiff's* hypothesis, a molecular agitation of the nerves and nervous centres, even granting that the mere neighborhood of metals or magnets could exert this action. On the other hand, there are perhaps other influences at work in all these experiments, not strictly physiological (in the usual sense) in character. These are influences of cerebral origin, though not necessarily such as are present to the mind of the patient. Of course, it need hardly be said that the possible influence of these agencies has been considered by all the best experimenters on this subject, and that steps have been taken to eliminate them. The question is, have these precautions been sufficient ? From the first there has been a strong party who have answered this question in the negative. This party includes those who believe, with *Carpenter* and *Tuke*, that the phenomena are those of "expectant attention," and those who refer them to the class of so-called "trance" phenomena (*Beard*), in which the involuntary, subconscious innovations play the most important part.

Until recently, *Beard* was a strong supporter of the view that this latter influence had not been sufficiently considered, and could not be until the sources of error in the usual methods of investigation had been more succinctly recognized. In his last publication, however, *Beard* admits that he cannot point out the flaws in the later experiments of *Charcot*, *McCall Anderson* and *Franz Müller*, the important condition being apparently fulfilled, "that the patient should be deceived all the time."

I am myself not prepared, without still further circumstantial evidence, of a kind which perhaps cannot be furnished till we learn more about this little-studied question of the interaction of the voluntary and involuntary life, to abandon *Beard's* first, strong position. Perhaps the systematic study of hypnotism, which is now being vigorously pursued, both in France and Germany, will throw more light on this kindred subject.

The investigation of these phenomena, while belonging both tc psychology and to physiology, has hitherto received but little systematic study from the representations of either of these sciences being rather shunned by each body as coming more properly under the cognizance of the other. It is verbally but not practically recognized, that in the acts of all of us, and not alone of the class of somnambulists, mesmerists, and hysterics, the involuntary life plays by far the most prominent part.

The lack of a full and practical recognition of this fact, that between hysterical and healthy persons there is not a gulf but a bridge, has led some of those who have written upon this subject to point to a few cases in which hemianæsthesia of organic, cerebral origin has likewise been relieved by metallo-therapeutics, as ruling out the mental influence which, in the case of the hysterical patients, might have been looked upon as the active agent.

The counter-criticism is important, but by no means so much so as has been maintained. It is evident that, even in these cases, the hemianæsthesia, though remotely of organic origin, is not so in the sense that all the conducting tracts between the periphery and the nervous centres have been severed, for in this case the recovery would involve slow and protracted nutritive changes, such as the formation of blood-vessels, multiplication of cells, absorptive processes, and the like; whereas, as *Vulpian* long ago showed, this anæsthesia may disappear quite rapidly under strong cutaneous faradization. The cause of its existence is, therefore, evidently a subtle one, possibly of an inhibitory character; or, more probably, due to a sluggishness, or inability, on the part of consciousness to recognize, without strong impulse from without or within, impressions coming through imperfect or unaccustomed channels.

However this may be, the pathology of organic cerebral anæsthesia is plainly still too obscure to justify positive a priori judgments as to what the nature of the remedy must be which should remove it.

DISEASES OF THE KIDNEY.

BY

A. B. BALL, M.D.,

ATTENDING PHYSICIAN TO ST. LUKE'S HOSPITAL,

OF NEW YORK.

ALBUMINURIA.

BIBLIOGRAPHY.

Maixner: Ueber das Vorkommen von Eiweisspeptonen in Harn. Vierteljahrschr. f. d. prakt. Heilk., 1875, XXXVI., 75.—Brunton, T. L., and D'Arcy Power: On the Albuminous Substances which Occur in Albuminuria. St. Bartholomew's Hosp. Rep., 1877.—Runeberg, J. W.: Die Filtration von Eiweisslösungen durch thierische Membranen. Arch. f. Heilk., 1877, XVIII., 1.—Ueber die pathogenet. Bedingungen der Albuminurie. Deut. Arch. f. klin. Med., 1879, XXIII., 41, 225.—Albuminurie bei gesunden Nieren. Arch. f. path. Anat., 1880, LXXX., 175.—Moxon, W.: On Chronic Intermittent Albuminuria. Guy's Hosp. Rep., 1878, XXIII., 233.—Rooke, T. M.: Note on Intermittent Albuminuria. Brit. Med. Journ., 1878, II., 596.—Griffith, G. de G.: On Reflex, Eccentric, or Irritative Albuminuria. Practitioner, London, 1878, XX., 13.—Dukes, C.: The Albuminuria of Adolescents. British Med. Journ., 1878, II., 794.—Leube, W.: Ueber die Ausscheidung von Eiweiss im Harn des gesunden Menschen. Virchow's Arch., 1878, LXXII., 145.—Nussbaum, M.: Fortgesetzte Untersuchungen über die Secretion der Niere. Pflüger's Arch. f. d. gesammte Physiologie der Menschen und der Thiere, 1878, XVII., 581.—Ueber die Entstehung der Albuminurie. Deutsch. Arch. f. klin. Med., 1879, XXIV., 248.—Johnson, Geo.: Latent Albuminuria: its Etiology and Pathology. Brit. Med. Journ., 1879, II., 928.—Fürbringer, P.: Zur Kenntniss der Albuminurie bei gesunden Nieren. Ztschr. f. klin. Med., 1879, I., 340.—Saundby, R.: The Diagnostic Value of Albuminuria. Brit. Med. Journ., 1879, I., 699.—Edlefsen: Mittheilungen für den Verein Schleswig-Holsteiner Aerzte. 1 Jahrg., No. 2, Aug. 1879.—Heidenhain, R.: Hermann's Handbuch der Physiologie, 1880, Bd. V., 367.

The study of the etiology of albuminuria and its various causes has recently received an important contribution from Drs. *Brunton* and *Power* (l. c.). *Bartels*, in *Ziemssen's* Cyclopædia (American edition, Vol. XV., p. 40), lays down the following proposition: "*In every case the explanation of the excretion of albumen by the kidneys must be sought for in disturbance of the processes by which the secretion of urine is effected.*" With this position—that albuminuria is always of *renal* origin—our authors take issue, and insist upon the importance of recognizing the occasional genesis of this symptom in the intestinal absorption of certain products of albuminous digestion, which are more readily transmissible through animal membranes than is the case with the ordinary form of albumen.

"The great differences which are observed in the behavior of albuminous urine when boiled and treated with nitric acid have led medical men to recognize that the albuminous bodies occurring in the urine are not always the same; that two or more kinds of albuminous bodies may sometimes be present in the

> urine at once. *Lehmann* (*C. J. Lehmann:* Zur Chemie des Eiweissharnes. Virchow's Arch., Bd. XXXVI., 125) showed that paraglobulin is generally present in albuminous urine along with serum-albumen, and his results were confirmed and extended by *Edlefsen* (*Edlefsen:* Beiträge zur Kenntniss der Eiweissstoffe des Harns. Arch. f. klin. Med., Bd. VII., 1870, S. 70) and *Senator* (*Senator:* Ueber die im Harn vorkommenden Eiweisskörper. Virch. Arch., Bd. LXX., 476). *Stockvis* demonstrated that albuminous bodies may be absorbed from the stomach and intestines, and excreted unchanged in the urine. Nor is it only undigested albuminous bodies which are thus absorbed and excreted. *Claude Bernard* observed that after partaking of a quantity of cooked eggs, his urine became albuminous. The coagulated albumen of cooked eggs could obviously not be absorbed without undergoing some previous change. But *Kühne* has found that the pancreatic juice, before converting coagulated bodies into peptones, seems to change coagulated bodies into something resembling their raw condition. In *Bernard's* observation, the cooked eggs which he swallowed seem to have undergone this change, and then been absorbed in the same way that raw eggs would have been. The fact that absorption of albuminous substances does take place from the intestines, makes it appear extraordinary that albumen is not more frequently found in the urine during digestion, and one can only suppose the reason to be, that it is only when the digestive powers are overtaxed or deranged, so as to digest the food partially, but not completely, that such an event occurs. In a clinical lecture published in the *Medical Times and Gazette* for April 10th, 1852, the late Dr. *Parkes* noticed that in cases of albuminuria the albumen was much increased after meals, and he ventured the hypothesis that the albumen was of a different quality, as well as increased in quantity. In the same journal, April 22d, 1854, he discussed the origin of this increase, and distinguished it by the name of food-albuminuria. This food-albuminuria he considered was not due to congestion of the kidneys during digestion, for the water of the urine is often diminished, and the solids do not increase in proportion to the albumen. He, therefore, thought it might be due to albumen not being altered in the stomach and liver, and, therefore, being eliminated like white of egg. He called attention to the fact that the antecedents of Bright's disease are often such as to impair the function of the stomach and liver, and that dyspeptic symptoms often appear before renal."
>
> "This subject was again taken up by Dr. *Pavy*, who confirmed *Parkes*' results regarding the increase of albumen in the urine during digestion, and tried to ascertain experimentally whether or not *Parkes*' supposition were correct, that the albumen found in the urine varied in character as well as in quantity at different times. It occurred to him its occasional presence in the urine might be due to its diffusibility being greater at one time than at another. He, therefore, employed a dialyzing apparatus to distinguish between the albumens, with the result of showing that very considerable differences exist in the readiness with which different specimens of albuminous urine pass through animal membranes."

As the method employed by Dr. *Pavy* was too troublesome for ready use, *Brunton* and *Power* adopted the coagulation-point as the simplest mode of distinguishing the different albumens. For this purpose a thermometer was held in the test-tube containing the urine, while heat was gently applied, and the temperature noted at which cloudiness began to appear.

The first series of experiments showed that the coagulating point of albumen varied considerably, not only in different cases of albuminuria, but also at different times in the same patient. The variations ranged from 144° F. to 180° F. In order to ascertain the extraneous influences which determined the coagulation-point of serum-albumen, they tried the effect of adding urea and uric acid to solutions of this substance, and found that the former raised, while the latter lowered the coagulation-point. It was evident, therefore, that the presence of urea in albuminous urine more or less counteracts the effect of uric acid and its salts, and *vice versa*, so that in any given case the temperature of the coagulation-point might be widely different from that at which the albumen would be precipitated from an aqueous solution. To obviate this difficulty, urine was diluted with water to a specific gravity of 1005, and it was found that this experiment sometimes lowered the coagulating point and sometimes raised it, but did not render it constant.

> "As the coagulating point of the albumen in urine varied considerably, notwithstanding our efforts to get rid of the effects of urea and neutral salts by dilution, it occurred to us that it would be advisable to ascertain whether the coagulating point of the soluble albuminous substances produced by the action of pancreatic juice upon solid albuminous bodies varied at different stages of the digestive process. For it is evident that, if this should be the case, and these soluble albuminous bodies should be absorbed into the blood and excreted by the kidneys in the same way as raw white of egg, they would cause the temperature at which coagulation occurred in the urine to vary, apart from any influence of urea, uric acid, or salts contained in it. In order to ascertain this point, fibrin obtained from pig's blood was digested with water and some pancreatine in a water-bath for about three hours, at a temperature of 100° to 105° F. until the fibrin had disappeared. The solution thus obtained is filtered, and after standing for twenty-four hours, the temperature of coagulation was observed."

Undiluted the solution coagulated in different experiments at 120°–134° F. Diluted with its own bulk of water, the solution coagulated at 136°–144° F. This variability was probably due to more or less digestion of the albumen by the pancreatine during the process of heating. Examination of the solution, after it had been standing forty-eight hours, showed the presence of a considerable amount of indol, and the coagulating point of the filtered solution had risen to 168° F. Another portion of fibrin was digested with pancreatine, and a portion of the solution, after an hour's digestion in a water-bath, showed a coagulating point of 146°–150° F. Another portion, after two hours and fifty minutes' digestion, coagulated at 138°–140° F. The effect of urea and uric acid upon the coagulating point of the products of pancreatic digestion was found to be the same as upon that of serum- and egg-albumen.

Numerous other experiments were made to ascertain the effect of food in causing albumen to appear in the urine; the effect of food in altering the coagulating point of the urine in cases of albuminuria; the presence of peptones, pepsin, and diastatic ferments; the albuminous substances in the urine which are derived from the blood, the coagulating point of the urine in different diseases, etc. The general results at which they arrived are summed up as follows:

> "There are various albuminous bodies which appear in the urine. Some of these are derived from the digestive canal, and others from the blood, of which they form ordinary constituents. Those derived from the digestive canal may be

either albuminous substances absorbed without undergoing digestion, as, for example, the white of raw eggs, soluble albuminous substances produced from coagulated albuminous bodies by incomplete digestion, or peptones. These albuminous bodies which form constituents of the blood, and which we have noticed in the blood, are paraglobulin and serum-albumen. The former is in comparatively small quantity, the latter forming the great bulk of the urine of ordinary albuminuria. . . . The effect of food is to increase the quantity of albumen in the urine, or even to make it appear when it is absent during fasting. Its effect on the coagulating point is not constant, although it generally lowers it. This may be due to the fact which we have observed, that, while the earlier products of digestion of fibrin have a lower coagulating point, the later products have one which is higher than that of serum-albumen. In connection with this point, it is interesting to notice that, while pepsin occurs normally in the urine, we failed to find it, possibly because we operated on too small a quantity, and nevertheless we obtained evidence of pancreatic ferment (trypsin). This is the first case we know in which the ferment has been found in the urine, and its loss in unusual quantity may possibly prove injurious to digestion, and be one cause why the albuminous products of imperfect digestion appear in the urine. Further observations are wanted, but this would seem to point to the existence of a vicious circle in this, as in other diseases, the albuminuria leading to the excretion of pancreatic ferment, and the loss of pancreatic ferment leading to imperfect digestion with excretion and loss of its soluble albuminous products. . . . It is certain that albuminuria cannot be removed, like diabetes, from its place among diseases of the kidney, to be classed entirely with diseases of disordered assimilation, and also in those cases where the kidney is undoubtedly diseased, the loss of albumen is increased by disordered assimilation. Attention to this point in the pathology of the disease will, of course, have an important bearing upon treatment. . . . The coagulating point of the albumen in urine may be readily ascertained without trouble at the bed-side. The indications which it gives are not decisive as the nature of the disease, but more extended observations may, and probably will, yield much information useful both in prognosis and treatment."

With respect to the *site of transudation* of albumen in the kidney, some recent experiments of *Nussbaum* (l. c.) are extremely interesting. In order to determine the point of albumen-transudation, he selected the og, as this animal is provided with a special arrangement of renal vessels, which permits the arrest of circulation in the glomeruli without interfering with the blood-current in the vessels supplying the urinary tubules. In amphibia the glomeruli derive their blood from the renal arteries, while the tubules are supplied by a system of wide-meshed capillaries fed by a renal-portal vein, and the vasa efferentia of the glomeruli. Ligation of the renal arteries in the frog, therefore, arrests the circulation in the vessels of the glomeruli, but substances injected into the general circulation (through the heart) still find their way into the kidney through the posterior extremities and wall of the trunk by means of the renal-portal vein, which collects the blood of these regions, and conducts it, with the blood of the vasa efferentia of the glomeruli, to the renal tubules and thence to the vena cava inferior. Substances injected into the heart of the frog after ligation of the renal arteries are consequently not found in the glomeruli, but are distinctly present in other blood-vessels of the kidney. It follows, therefore, that if the urine be examined in permeable and occluded conditions of the renal arteries, the location where the excretion of injected substances takes place can be positively determined. *Nussbaum* found that, if the renal arteries of a frog were ligated, and two different substances injected into the general circulation, viz., a mixture of equal parts of fluid albumen from a hen's egg, or a ten per cent solution of peptone and a ten per cent solution of urea, the urine remained free from albumen; on the other hand, the same injection, without ligagation, produced albuminuria (0.5 ccm.). That some alteration occurs in the vessels of the glomeruli as a condition of transudation of albuminous substances was shown by the fact that, while, after ligation, no albuminuria followed an injection of a ten-per-cent solution of urea, albumen appeared in the urine for a longer or shorter time as soon as the ligatures were loosened.

As regards *the conditions of filtration of albumen through the vessels of the glomeruli*, *Runeberg* (l. c.) opposes the common view that the transudation is favored by *high* pressure in the vessels, and holds that the results of numerous experiments made by him clearly demonstrate that a *low* pressure is the essential condition. The filtration membranes used were dog, sheep, and rabbits' intestines, which had been removed from the animals immediately after death, carefully separated from the mesentery, thoroughly washed with water or a dilute solution of common salt, and then kept in dilute alcohol. Before their employment, the intestines were washed with distilled water, or a very dilute solution of common salt, and saturated with the experiment-fluid. The experiments gave the following results:

(1) When a fresh membrane was used, or one that had not been subjected to pressure for several hours, the relative amount of albumen in the filtrate diminished in proportion to the duration of the filtration, up to a certain point, and then remained relatively constant so long as the pressure continued the same.

(2) When this constant rate of filtration was reached, the amount of albumen in the filtrate diminished on increasing the pressure, and increased when the pressure was lowered.

(3) The amount of albumen in the filtrate, with equal pressures, was considerably greater when the membrane had for some time previously been free from pressure, than when it had been long subjected to pressure.

(4) Very finely divided emulsions, whose suspended particles are able to pass through the intestinal wall, present the same phenomena of filtration as albuminous solutions.

(5) The relative amount of albumen in the filtrate, other conditions being the same, varies considerably, according to the nature of the albuminous fluid, that is, according to the modification of albumen contained in it. The amount is greatest with solutions of egg-albumen, the filtrate often containing as large a quantity as the original solution; much less in solutions of serum-albumen, and least with casein-solution, such as is present in cow's milk. It is also somewhat greater in solutions which have once been filtered through a membrane.

(6) The rate of filtration, other conditions being the same, varies materially with the nature of the solution. In saline solutions it is much greater than in solutions of albumen, and noticeable differences obtain with different albuminous and saline solutions. Thus, for example, a solution of common salt filters much more rapidly than a solution of carbonate of soda; and if these salts be added to albuminous solutions, the common salt accelerates, and the carbonate of soda diminishes the filtration rate of the albumen.

In a later article (l. c.) *Runeberg* applies these experiments to the interpretation of the pathogenesis of albuminuria. It will be impossible here to follow him through the whole course of his extended argument, but at least a brief summary of his views seems desirable, inasmuch as their acceptance would involve a radical reconstruction of at least a part of the commonly accepted doctrine of albuminuria. In the first place, he holds that his experiments demonstrate the following propositions: (*a*) that the so-called albuminous solutions are merely more or less finely divided emulsions, the albumen particles of which possess different degrees of diffusibility, according to the kind of albumen present; (*b*) that animal membranes change their permeability for albumen molecules with varying degrees of pressure, becoming less permeable with a high degree, and more permeable with a low degree of pressure, and (*c*) that where fluid is present upon both sides of a membrane, and the pressure is unequal on the two sides, the difference between the two pressures produces the same effect as a unilateral pressure, that is, a diminution of the upper pressure as compared with the lower increases the permeability of the membrane, and *vice versa*. Thus the membrane may become more permeable to albumen, not only from a positive lowering of the upper pressure, but also from a relative increase of the counter-pressure.

Now, it is to be noticed that *Runeberg's* observations accord strikingly with certain experiments made upon *living* animals. Temporary ligation or compression of the renal arteries or of the aorta at a point above their origin has always been found to produce albuminuria (*Munk*, *Stokvis*, *Hermann*, etc.) *Hermann* attempts to explain this fact by supposing that during the arrest of circulation the blood-corpuscles accumulate in the interstitial capillaries so as to form an obstruction to the restored current, and thus raise the pressure in the glomeruli. The assumption is wholly gratuitous, as it rests on no direct observation, and is further opposed by the fact that albumen appears in the urine even when the flow of blood through the renal vessels is merely diminished without being entirely checked. That a lowered blood-pressure in the glomeruli is the true explanation of the albuminuria following these experiments *Runeberg* regards as probable, from the recurrence of the same symptom in cholera and violent attacks of diarrhœa, with vomiting, at stages in these affections when there can be no question that the tension in the glomeruli is much diminished by the excessive loss of fluid and depressed action of the heart.

On the other hand, the demonstrable presence of increased pressure in the vessels of the glomeruli is not followed by filtration of albumen. Thus *Stokvis* and others have shown that when the blood pressure is increased in the renal arteries, either by ligating the aorta below their origin, or by extirpating one kidney, no albuminuria is produced so long as the pressure is not sufficiently great to rupture the renal vessels and permit admixture of blood with the urine. Nor does albuminuria result in those forms of heart-disease which are attended by increased arterial tension—such as aortic insufficiency—until the blood-pressure is lowered by degeneration of the cardiac muscle, and then the albumen in the urine is generally found to increase in proportion to the progressing enfeeblement of the heart.

Furthermore, the conditions which give rise to albuminuria in connection with obstruction to the *venous* circulation in the kidneys will be found, on careful analysis, to favor rather than oppose *Runeberg's* views, It is well known (*Robinson*, *Meyer*, *Frerichs*, *Munk*, *Stokvis*, and others) that ligation or constriction of the renal veins, or of the inferior vena cava above the entrance of the renal veins, is followed by albuminuria in the lower animals. At first sight, this obstruction to the outflow of blood from the kidneys would seem to favor the transudation of albumen by raising the blood-pressure in the vessels of the glomeruli, but there are several considerations which oppose such an explanation. In the first place, veins in general are much more capacious and elastic than arteries, and may therefore undergo considerable distention without increasing the backward pressure. Again, the Malpighian vessels are more fully protected from the effects of backward pressure from the veins than is the case with any other capillaries in the entire body. Between the veins and the vessels of the glomeruli intervene the interstitial capillary system and the vasa efferentia, while a portion of the branches of the renal artery pass directly into the interstitial capillaries without previously forming glomeruli. In view of this peculiar arrangement of the renal vessels, many writers (*Bartels* and others) have supposed that, in such cases, the albumen transudes, not from the vessels of the glomeruli, but from the capillary network surrounding the renal tubules, where the blood-pressure would naturally be greater than in the vessels of the glomeruli. It is to be noted, however, that the interstitial capillaries are not in immediate contact with the renal tubules, but are separated from them by lymph spaces, which receive any transudation from the capillaries and transmit it to the lymphatics of the kidney. In order to mix with the urine, therefore, a transudate from the interstitial capillaries would have to pass through the membrana propria of the tubules as well as its epithelial lining. In the absence of direct proof that the conditions in venous obstruction favor such a double filtration of albumen, this theory must be abandoned, and we must fall back upon the glomeruli as the site of transudation. In the analogous conditions which arise in connection with the obstructed venous circulation of certain forms of heart disease, there are disturbances of circulation which point clearly to a lowered pressure in the vessels of the glomeruli as a determining cause of albuminuria. Under these circumstances, albumen does not appear in the urine until the general arterial tension, and consequently that in the glomeruli, is lowered by failure in the heart's action, while remedies which directly tend to increase arterial tension, at the same time diminish or arrest the the albuminuria.

As regards *febrile* albuminuria, *Runeberg* rejects the commonly accepted interpretation that the symptom is due to paralysis or relaxation of the renal capillaries arising from elevated temperature. Albumen, he urges, may appear in the urine when the temperature is low, and may be absent when the latter is quite high. On the other hand, mere dilatation of the arterial vessels does not produce albuminuria. After section of the vaso-motor nerves of the kidneys, no albuminuria results so long as the blood-vessels are not compressed. It is to be noted, moreover, that, besides the elevated temperature, there is a group of symptoms common to all severe and continued febrile diseases, which affords a more rational explanation of the albuminuria. Disturbances of circulation pointing to lowered arterial tension are constantly present under these circumstances, arising not only from feeble action of the heart, in consequence of the paralyzing influence of elevated temperature and possibly parenchymatous degeneration of the cardiac muscle, but also from general paralysis of small arteries and capillaries throughout the body.

The interesting cases of albuminuria after cold bathing reported by *Johnson* (*Brit. Med. Jour.*, 1873, p. 664) and ascribed by him to contraction of the cutaneous vessels and congestion of the kidneys with increased pressure in the Malpighian vessels, *Runeberg* interprets in a different way. A considerable general cooling of the body has been shown, by experiments upon the lower animals, to depress both the cardiac and respiratory functions, the breathing becomes superficial, the heart beats feebly, and the arterial tension is lowered throughout the body, and consequently in the vessels of the glomeruli.

Passing over, for want of space, the application of *Runeberg's* theory to the interpretation of the albuminuria produced by cantharides, turpentine, lead, iodine, and other poisons, we notice, in conclusion, the support his views receive from the beautiful investigations of *Cohnheim* on embolic processes and inflammation (Neue Untersuchungen über die Entzündung). *Cohnheim* has shown incontestably that the extravasation of the formed elements of the blood in inflammatory processes is not due to increased pressure in the capillaries, nor to retarded circulation in the dilated veins, nor to co-operation of both these conditions. If the veins be ligated and at the same time the vaso-motor nerves running to the same vascular district be divided, there filters through the walls of the vessels only the usual serous transudate. There is no trace of *inflammatory* exudation, that is, of exudation containing large amounts of albumen and fibrin, and the white corpuscles which escape are few in number. Now, if the blood-flow to the vascular district be checked by compressing the artery leading to it, so as to depress the normal blood-pressure upon the walls of the vessels, the permeability of the latter is thereby increased. With the restoration of the circulation, a more albuminous transudation takes place, accompanied by diapedesis of large amounts of the formed elements of the blood.

Similar phenomena ensue when an embolus in a terminal artery cuts off the flow of blood to a vascular district, and thus, for a time, depresses the lateral pressure in the vessels anterior to the point where the embolus is situated. The vessels, freed from the influence of arterial pressure, gradually fill with blood from the veins, and, as a result of the diminished pressure, the walls of the vessels now permit the formed elements of the blood to pass out and form the so-called hemorrhagic infarction. If the normal circulation be restored before important nutritive disturbances have taken place in the walls of the vessels, the latter resume their usual density, and the abnormal filtration ceases.

The forms of albuminuria thus far considered, *Runeberg* holds, are caused chiefly, at least, by increased permeability of the vessels of the glomeruli, due to diminished pressure, but besides these forms there are others in which the albuminuria is directly dependent on inflammatory or degenerative changes in the vascular membrane. Under such circumstances, the filtration of albumen may take place even when the blood-pressure in the Malpighian vessels is above the normal, although even here conditions of blood-pressure operate in the same direction as in a normal state of the vascular membrane. Thus, in granular kidney, where a chronic inflammatory process similar to that in the interstitial tissue is very constantly present in the vessels of the glomeruli, the increased arterial tension and polyuria may be accompanied by the transudation of a small amount of albumen, but, as a rule, no large quantity escapes until towards the end of the disease, when the blood-pressure is lowered by degeneration of the cardiac muscle.

During the present year *Runeberg's* views have been subjected to a searching criticism by *Heidenhain* in *Hermann's* Physiology (l. c.). He shows, by an analysis of the results of *Runeberg's* experiments, that, while the *percentage* of albumen in the filtrate diminishes with increased pressure, the *absolute* quantity of albumen is actually increased. In the following table *Heidenhain* has added to *Runeberg's* figures estimates of the absolute amounts of albumen filtered.

	Pressure in cm. water height.	Amount of filtrate in ccm. per hour.	Percentage of albumen in filtrate.	Absolute amounts of albumen in ccm. p. hour.	REMARKS.
1	100	472	8	37.76	Beginning of experiment.
2	100	90	6.54	5.88	After three hours of equal pressure on the membrane.
3	10	24	7.8	1.87	After two hours of diminished pressure.
4	10	14	6.84	0.95	On the morning following a night with same pressure.
5	40	25	5.2	1.30	
6	100	30	3.84	1.15	
7	100	29	3.88	1.12	On the following morning.
8	40	16	4.52	0.72	
9	10	8	6.54	0.52	

On comparison of 1 and 2, or 3 and 4, it is clear that *Runeberg* is correct in asserting that with a constant pressure the membrance becomes gradually less permeable to water and in a still higher degree to albumen. On the other hand, if we compare 4, 5, 6, or 7, 8, 9, a reference to the column exhibiting the *absolute* amounts of albumen will show that *more* albumen is filtered with a *high* than with a *low* pressure. *Runeberg's* mistake arose from estimating solely the *percentage* of albumen in the filtrate. With increased pressure both more albumen and more water are filtered, but the albumen-stream increases more slowly than the water-stream, so that the *percentage* of albumen in the filtrate relatively decreases with heightened pressure.

Furthermore *Heidenhain* regards *Runeberg's* position that the albuminuria produced by temporary constriction or closure of the renal artery is due to diminished pressure within the glomeruli as wholly untenable for several reasons. In the first place, it has just been shown that in *Runeberg's* experiments the albumen-filtrate diminished with decreasing pressure. Again, the membranes used by *Runeberg* differ in their filtration-properties from the walls of the glomeruli, as appears from the fact that the former readily filter hæmoglobin, while, according to *Ponfick*, this substance does not escape from the glomeruli, but is discharged by the epithelium of the renal tubules. Thirdly, the increased permeability to water and albumen, which *Runeberg* observed in animal membranes after pressure had been withdrawn for some time from the latter, is not noticed in the living kidney. For, after the renal artery is reopened, the glomeruli are at first impermeable to water; the secretion is arrested and may remain so for three-quarters of an hour. When re-established, the secretion gradually increases in amount, of course only up to a certain limit. In the filtration experiments, on the other hand, the amount of filtrate is greatest at the outset, and steadily diminishes. There is, therefore, a fundamental difference between the physical experiment and the physiological process.

Equally unsatisfactory, in *Heidenhain's* opinion, is *Runeberg's* explanation of the albuminuria which results from obstruction to the return flow of blood through the renal veins. *Runeberg*, it will be remembered, holds that in this case the filtration of albumen depends upon a *relative* decrease of pressure in the glomeruli as compared with the pressure in the tubules, the latter pressure being raised by the compression of the tubules by the distended renal veins. Whatever may be the pressure-condition in the tubules, an *absolute* decrease of pressure in the *glomeruli* is out of the question, and it is hardly probable that a *relative* lowering of pressure can operate in an opposite direction to that obtaining with an absolute diminution.

Heidenhain suggests, in conclusion, that the filtration of albumen may possibly depend upon a condition common to both arterial and venous obstruction, viz., slowing of the blood-stream. The normal secretion of urine is clearly dependent upon the maintenance of a normal rapidity of circulation. The epithelial cells in the glomeruli are extremely susceptible to even temporary deprivations of blood, and it is not unreasonable to suppose that between the condition in which they perform their function normally, and that in which their function is entirely arrested, there may be an intermediate stage which permits the discharge of an albuminous instead of the normal secretion.

Within the last twenty years, especially within the last decade, a remarkable change of opinion has taken place with respect to the value of albuminuria *per se* as a sign of renal disease. Whereas formerly the detection of albumen in the urine was regarded as almost a fatal omen, it is now even seriously debated whether albuminuria may not in certain cases fall within the limit of physiological conditions.

During a discussion at the Royal Medical and Chirurgical Society of London in 1873 (*Lancet*, 1873, 1, 808) on a paper by Dr. *Geo. Johnson* analyzing 200 cases of albuminuria, Sir *Wm. Gull* asked "what worth mere statistics of albuminuria had, since it occurred in young and growing men and boys, almost as frequently as spermatorrhœa?" This suggestion, derived from Sir *Wm. Gull's* habit of examining the urine in all cases of disease, has been more fully elaborated by several recent writers. Dr. *Moxon* (l. c.) calls attention to the fact that young men not infrequently suffer from an intermittent albuminuria, which continues for a considerable period in an irregular way, and finally disappears completely without leaving any evident traces of structural changes in the kidneys. He confines his remarks to this condition in young men, because he has had no opportunity for observing it in female patients. This form of albuminuria he calls the *albuminuria of adolescents*, as it seems to be connected in some way with the period of adolescence, and has not been met with by him at other periods of life. The nineteen cases of which he possesses notes, bore a sufficient resemblance to each other in their symptomatology to justify their grouping into a common class. The symptoms were generally of an indefinite character: languor, unrefreshing sleep, anæmia, perhaps headache, and indisposition to social intercourse. The patient himself made light of his complaint, and medical advice was sought rather to relieve the anxiety of relatives or friends. A physical examination of the various organs failed to disclose organic disease, and the cases might naturally have been dismissed as due to simple debility, had not albumen been detected in the urine. The albuminuria was intermittent, making it necessary to examine the urine for two or three days consecutively, as well as portions secreted at different times of the day, as albumen was often present at one time of day and not at another. The urine passed after breakfast was most frequently found to contain albumen. No casts were found, except in a few cases at rare intervals, but there were commonly numerous crystals of oxalate of lime. The previous occurrence of scarlet fever or diphtheritic sore throat was carefully excluded. In all of the cases in which the subsequent history could be traced, the albuminuria, after running an irregular course with numerous fluctuations, finally disappeared permanently.

Dr. *Clement Dukes* (l. c.) has also repeatedly noticed the occurrence of albuminuria in growing boys under conditions similar to the above. The presence of albumen in his cases was generally intermittent and transitory, although for months the albuminuria was apt to return with every chill or error in diet. In other instances it was very persistent, except when the patient was kept in bed and put upon a milk diet. "This albuminuria of puberty," he says in conclusion, "is often so persistent, and, even when it has disappeared, recurs again and again so often, that I fear many of the cases pass on to chronic Bright's disease, although this I can only surmise, as I lose sight of them, but long-continued or often repeated hyperæmia is the cause of all kidney diseases."

Dr. *Rooke* (l. c.) has recorded a similar experience in young women or rather growing girls between the ages of fourteen and sixteen, who were suffering from anæmia with a *bruit de diable* in the cervical veins. He calls special attention to one circumstance which may perhaps throw some light upon the pathogenesis of the albuminuria, viz., that in his cases albuminuria could be made to appear and disappear according as the patient was allowed to walk about or was kept strictly in bed.

Dr. *Saundby* (l. c.) reports the results of examinations of the urine in one hundred and forty-five male patients taken *seriatim* as they presented themselves before him in the out-patient department of the General Hospital in Birmingham, England. Of this number no less than one hundred and five contained albumen in more or less quantity, easily detected by boiling and adding a drop or two of acetic acid. Sixty-six of the cases were tabulated as granular kidney and one as fatty kidney or chronic parenchymatous nephritis. The number of cases of granular kidney, he says, seems a large porportion, but the symptoms were carefully investigated before a diagnosis was made, and the proportion (sixty-six to a total of three hundred and fifty men, women, and children) does not exceed the *post-mortem* room statistics of the general hospital. It is not easy to see upon what grounds Dr. *Saundby* ascribes such a large proportion of the cases of albuminuria to granular kidney, when we consider that albuminuria is far from being a constant symptom in this affection, and that, even admitting his diagnoses to be correct, the chances were very great against his finding albumen in all the cases of renal cirrhosis. Many of the other cases resembled clinically the types described by Drs. *Moxon* and *Dukes*, while in still other instances there was nothing characteristic, and the patients were to all appearance healthy young men suffering from a merely temporary derangement. The microscopical examination was usually negative; occasionally a few oxalates were found, more rarely a few hyaline casts, which, as is now generally admitted, have no pathological significance. Gonorrhœa as a source of albuminuria was carefully excluded. Spermatorrhœa could not account for the albuminuria in any cases, for the seminal and prostatic fluids do not contain any albuminous body coagulable by heat (*Simon*). The remarkable proportion of cases of albuminuria found by Dr. *Saundby* must certainly be regarded as unique, and whatever its explanation may be, it is difficult to derive from his cases any conclusion except that albuminuria is *per se* no indication of renal disease.

An interesting contribution to the subject of *physiological* albuminuria has recently been made by *Leube* (l. c.) His attention was first drawn to this question in the winter of 1877, by finding albumen in the urine of several patients whose condition did not warrant the least suspicion of renal disease. The amount of albumen was so small that it could be detected only by comparing the tested urine (heat and nitric acid) with an untested specimen against a dark background. Supposing that albuminuria was under all circumstances to be regarded as a pathological phenomenon, he diagnosticated in all the cases a latent affection of the kidneys. The appearance of a slight turbidity in the urine occurred too frequently, however, in patients without any symptoms of renal disease to warrant such a diagnosis, and he therefore undertook an extended series of experiments upon one hundred and nineteen soldiers belonging to a battalion stationed in Erlangen, in order to ascertain whether traces of albumen in the urine might not to a certain extent be regarded as a normal condition. The examinations were repeated for seven days and embraced both the morning urine, *i. e.*, the urine passed immediately after rising, and that passed at mid-day. The latter represented the effects of exercise —a march of about five hours, or several hours' drill, during the months of June, July, and August, with a temperature of from 55° to 77° F.

The method adopted for the detection of albumen was the following: The freshly passed urine was first filtered. A specimen was then boiled in a test tube, and treated with nitric acid. After a second boiling it was compared with an unboiled specimen of the same urine against a black surface. If any turbity showed itself in the boiled urine, the specimen was partially evaporated, treated with two drops of acetic acid, and the deposit allowed to subside. The supernatant fluid was then decanted and replaced by water. This procedure was repeated until the supernatant fluid was as colorless as possible. The fluid and precipitate were then neutralized, and the precipitate was washed upon a filter. One portion of the washed precipitate was tested with *Millon's* reagent, and another specimen boiled with caustic potash ; the cold solution was then treated with two drops of a dilute solution of sulphate of copper. If a positive result followed these tests, a purple-red color with *Millon's* reaction and a violet color—generally, of course, only feebly indicated, but sometimes an intense red-violet—with the caustic potash test, the urine was regarded as containing albumen. The general result of these examinations was as follows:

Morning urine albuminous in 5 out of 119 soldiers, .	4.2 per ct.
Mid-day " " " 19 " " " " .	16.0 " "
Mid-day alone (morning urine free from albumen), in 14 out of 119 soldiers, ...	11.8 " "
Mid-day and morning urine both albuminous in 5 out of 119 soldiers, ...	4.2 " "

An attempt was made to ascertain the kind of albumen present in these cases, but the amounts were too small for satisfactory analysis. No casts or blood-corpuscles were found in the sediment. This physiological albuminuria was characterized by the scanty amount of albumen which appeared in the urine. Although varying in different cases from scarcely perceptible traces up to complete opacity of the boiled urine, it was still, on the whole, insignificant in amount, and certainly did not exceed 0.1 per cent. All the soldiers in whose urine albumen was found were carefully examined for gonorrhœa and hypertrophy of the heart, but without the detection of these or any other morbid condition. The very temporary nature of the albuminuria was strikingly shown in one soldier whose urine on two different days had been free from albumen both in the morning and at evening, but contained albumen on another day at 10 A.M., after a four hours' drill, while the urine passed between 4 and 6 P.M. of the same day was perfectly normal.

A further contribution to this subject has been made by *Edlefsen* (l. c.). He reports three cases of albuminuria, independent of renal disease, occurring in anæmic individuals after exertion, the urine during rest being always free from albumen. In accordance with *Runeberg's* filtration-laws, *Edlefsen* explains such cases by the diminished blood-pressure within the glomeruli during active exercise. Not only does the increased flux of blood to the muscles at such times lessen the flow to other organs, and consequently to the glomeruli—thereby lowering the blood-pressure within the vessels of the latter—but the counter-pressure in the urine-canals is indirectly raised in the following way: In anæmic persons, muscular exertion produces excessively accelerated action of the enfeebled cardiac muscle, and blood accumulates in the lesser circulation. General venous congestion ensues, the renal veins become distended, and by narrowing the urine-canals impede the outflow of urine, thus increasing its backward pressure upon the filtration-membrane. *Edlefsen* also advances an ingenious hypothesis in explanation of *Runeberg's* law of filtration. He suggests that the pore-canals of membranes, instead of passing through perpendicularly to the surface, may do so *obliquely*, or in such a way as to be compressed by increased pressure, and to become more patent as the pressure diminishes; thus either hindering or promoting the escape of emulsion-particles of albumen, according to the degree of pressure. As *Fürbringer* has pointed out, however, this hypothesis necessitates the existence of another set of pores for the passage of the water of the urine, since in albuminuria the filtration of albumen and that of water usually proceed in an inverse ratio; but this difficulty, so far as we can see, equally attends any conceivable arrangement of filtration-pores.

In an article on "*albuminuria with healthy kidneys*" *Fürbringer* (l. c.) reports fourteen cases similar, in many particulars, to those already described. One of the cases, however, presents some points of special interest.

A physician, 29 years of age, in good health, after an hour of great anxiety early in the morning, passed urine containing a considerable amount of albumen. The following urine showed less albumen, and that passed in the evening, when the mental depression had passed off, was entirely free from albumen. During the following week, the urine averaged 1100 to 1640 ccm., specific gravity 1018 to 1022, and occasional traces of albumen were detected. After a second profound depression of spirits from reception of bad news and great fear for the safety of relatives, he passed a *small* quantity of dark, clear, strongly acid urine, of a specific gravity of 1030, and containing a large amount of albumen (0.31 per cent). Uric acid crystals were found in abundance, but no elements pointing to nephritis. The same symptoms recurred on two subsequent occasions of great alarm and depression of spirits. Each attack was accompanied by a dull sensation of pain over the region of the kidneys, lasting for several hours, and accompanied by marked sensitiveness to percussion in this locality. The quantity of urine at such times was always *below* the normal, and increased to a marked extent as the mental depression wore away. Traces of albumen were found at intervals for nearly eight months, when the symptom entirely disappeared. No evidences of anatomical lesion were found at any time. There were never any blood-corpuscles or granular casts. In the albuminous specimens, uric acid or oxalate of lime, or both together, mixed with amorphous urates were commonly present, and occasionally small, perfectly hyaline and pale casts were found, but these, as is well known, show nothing in favor of renal disease. Neither gentle exercise nor long walks appeared to influence the transudation of albumen, but *severe* exertion almost always occasioned albuminuria. No effect was noticed from diet. Neither eggs in large number, nor stimulating food, strong spices, liquors produced any such result. On the other hand, the albuminuria, when present, was always diminished or entirely checked by ingestion of large quantities of fluids, if the latter had the effect of largely increasing the urinary secretion. The result was the same, whatever the nature of the fluids, whether water, soup, beer, or coffee. It was very noticeable, however, that no diuresis occurred from copious drinking immediately after the attacks of mental depression previously referred to.

The influence of depressing emotions in the production of albuminuria *Fürbringer* explains by the general arterial ischæmia (and therefore presumably lowered tension in the vessels of the glomeruli), which accompanies profound emotional disturbances of a depressing character, such as fright, terror, etc. The sunken countenance, bloodless skin and mucous membranes, and almost imperceptible pulse frequently noticed under such circumstances point to reflex irritation of the cardiac nerves, proceeding from excitation of the reflex inhibitory centres, as well as to a general arterial spasm. Emptiness of the arteries involves accumulation of blood in the venous system, and, as has been shown in the analogous case of surgical shock, such venous accumulation probably takes place chiefly in the large reservoir of the cœliac, mesenteric, and renal veins with their numerous branches. Acute cyanosis of the kidneys is therefore present with the conditions demanded by *Runeberg* for the establishment of albuminuria, viz., diminished pressure in the glomeruli, and increased backward pressure in the urine-canals from compression by distended veins. The painful sensation in the renal regions may be referred to distention of the capsules of the kidneys produced by acute swelling of the organs.

Besides the case just described, *Fürbringer* reports thirteen other cases of intermittent albuminuria without renal disease. Seven of these were observed while conducting an examination with reference to this point upon sixty-one children (from three to six years of age) belonging to a child's protectory. At this early age, the existence of granular kidney or admixture of sperm-plasma with the urine was naturally out of the question, while the habits of life, especially as regards food and exercise, could be easily investigated in full detail. Observations were continued through August, September, and October of 1879, that is, for the positive cases. In three of the seven cases of albuminuria, albumen was detected only once; of the other four, one showed albumen only twice, while in the remaining three intermittent albuminuria continued for weeks or months, but without any elements in the urine pointing to nephritis. It was noticed that, with very few exceptions, the albumen appeared only in the urine of the later hours of the *forenoon*, after hard play during fasting. In the early morning and late in the evening, albumen was always absent when sought for.

While accepting, in general, the results of *Runeberg's* experiments, *Fürbringer* maintains that diminished pressure in the vessels of the glomeruli is not the whole explanation of "albuminuria with healthy kidneys." Albuminuria does not necessarily occur even when the blood-pressure is considerably diminished. He inclines, therefore, to *Leube's* hypothesis of an *individual permeability* of membrane, but confesses his inability to explain such differences in permeability. Possibly temporary derangements of innervation within the kidney may be a factor in these cases, and he suggests, finally, that some light may be thrown upon the subject by experiments in which the condition of the urine is observed when the pressure in the vessels of the glomeruli has been lowered by abstraction of blood, arterial compression, etc., while the renal nerves are at one time allowed to remain intact and at another irritated or divided.

In this connection may be mentioned a case of *reflex* albuminuria reported by Dr. *Griffith* (l. c.). The patient was operated on for anal fistula, the wound being dressed in the usual way. A few days afterwards, partial suppression of urine occurred, with uræmic symptoms, and on examination a large amount of albumen was found in the urine. The dressings were discontinued, and a free purge given, followed by a diuretic. The albuminuria and uræmic symptoms now disappeared, but returned again as before when the dressings were resumed. A second time the symptons yielded to a discontinuance of the dressings. A simple unirritating treatment was now adopted; the wound healed kindly, and for the past eight years the patient has enjoyed good health, without any recurrence of the renal symptoms. Several cases similar to the above have occurred in Dr. *Griffith's* practice—one in which the patient died from the supervention of uræmia of apparently reflex origin—and he is confident that the production of albuminuria and uræmia by operations in the neighborhood of the genito-urinary tract is more frequent than is commonly supposed.

Dr. *Geo. Johnson*, in a recent article on "latent albuminuria" (l. c.), maintains that albuminuria, even in its least marked forms, is always pathological, never physiological, and that the latent affection can be traced in a very large proportion of cases to some definite cause. In some of these cases, careful inquiry will disclose a previous history of acute nephritis, dating back perhaps for months or years, and resulting from exposure to cold or wet, or from an attack of scarlet fever, measles, diphtheria, erysipelas, typhus and typhoid, pyæmia, rheumatic fever, etc. In other instances, although there may have been no previous illness to explain the renal disorder, the albuminuria may perhaps be traced, especially in boys and young men, to chilling of the surface of the body by perspiration induced by active exercise. The same result occasionally follows cold bathing. In still another class of cases, the presence of albumen in the urine may be due to imperfect digestion of albuminous food and elimination of modified albumen, or to a diminished tone and contractile power in the muscular walls of the arterioles, as an accompaniment of the general nervous exhaustion often seen in cases of chronic dyspepsia. Such a loss of tone in the nervous system, with imperfect digestion of albuminous substances, may be due in some instances to excessive tobacco-smoking, occasionally also to the free use of alcoholic stimulants, and not infrequently to mental anxiety. The latter cause probably operates here, as in the production of diabetes, primarily through its influence upon the chylopoietic viscera, especially the liver. Whatever may be the explanation for individual cases, *Johnson* regards the albuminuria as always important, and as threatening, if neglected, an ultimately fatal disorganization of the kidneys by the conversion of the temporary and occasional into a persistent albuminuria.

The problems here presented with respect to the genesis and prognostic significance of latent forms of albuminuria can, of course, be solved only by the results of future investigation, but while all must agree with *Johnson* that the symptom should always be closely watched, his dogmatic assertion that the presence of albumen in the urine is invariably pathological seems somewhat premature. No one can dispute his proposition if all kidneys are to be tried by an ideal standard of absolute impermeability to albumen on the part of their membranes, but may not a leakage of this kind be to a certain extent *physiological* in some individuals as a result of severe exercise, posture, and other conditions ? The observations of *Leube*, *Fürbringer*, and others seem to point in this direction, at least so far as to indicate the occasional presence of an individual permeability which can hardly be termed pathological. Dr. *Clement Dukes* refers the *albuminuria of adolescence* to a physiological increase of arterial tension at this period, similar in its nature to the increased vascular pressure which *Mahomed* has shown to exist during pregnancy. This opinion, however, is unsupported by sphygmographic evidence, and is advanced simply as a plausible conjecture. The whole question of latent albuminuria is one of extreme interest, and it is to be hoped that its investigation will be prosecuted until some reliable conclusions can be drawn.

URÆMIA.

BIBLIOGRAPHY.

Strumpell, A.: Bemerkungen über die Urämie und ihren Einfluss auf die Körpertemperatur beim primären Morbus Brightii. Arch. d. Heilk., 1867, XVII., 36.—**Mahomed, F. A.**: On the Pathology of Uræmia and the so-called Uræmic Convulsions. Brit. Med. Journ., 1877, II., 10, 42.—**Cuffer, P.**: Recherches cliniques et expérimentales sur les altérations du sang dans l'urémie et sur la pathogénie des accidents urémiques. De la réspiration de Cheyne-Stokes dans l'urémie. Paris, 1878.—**Perrineau, L.**: Contribution à l'étude de la dyspnée urémique. Paris, 1879.—**Ortille, E.**: De la dyspnée nerveuse des nephrites: état des gas du sang chez les urémiques. Lille, 1879.—**Persoons, H.**: Néphrite albumineuse, etc.; considerations à propos de la pathogénie des phémonènes dits urémiques dans la néphrite albumineuse. Presse méd. belge, 1879, XXXI., 57, 65, 73, 81, 89.—**Chambige, L.**: Etude sur la mort subite dans l'urémie. Paris, 1879.—**Morat** et **Ortille**: Recherches sur les alterations du sang dans l'urémie. Comp. rend. Acad. d. Sc., Paris, 1879, LXXXVIII., 1035, 37.—**Stolnikow**: Die Temperatur herabsetzende Wirkung chronischer Nierenentzündungen. St. Petersb. Med. Wochschr., 1879, IV., 416.—**McBride, T. A.**: The Temperature in Uræmia. Archives of Med., N. Y., 1880; 1, 65.

In an article "On the pathology of uræmia and the so-called uræmic convulsions" (l. c.), Dr. *Mahomed* calls attention to a cerebral lesion which, in his opinion, accounts for the epileptiform convulsions generally known as uræmic, viz., the presence of numerous punctiform hemorrhages in the gray matter of the cerebral convolutions. These minute extravasations were found by him in several cases of Bright's disease, in which death was preceded by convulsions and coma, and microscopic examination showed that the hemorrhagic points were true hemorrhages, and not merely transversely-divided sacs of the miliary aneurisms described by *Charcot* and *Bouchard*. That these hemorrhages were not the *result* of the convulsions *Mahomed* infers from the absence of such extravasations in epileptic convulsions, all authorities agreeing that such a lesion is very rare in epilepsy. The infrequency of similar observations in Bright's disease may be explained partly by the fact that the head is rarely examined in this affection, partly by the rapid absorption of these small extravasations, rendering their discovery improbable if the convulsions have not occurred within a few days before death, and partly also by the great difficulty of exploring *all* the gray matter of the convolutions. Now, however, that we are able to locate the motor areas of the convolutions with some degree of accuracy, he is confident that these punctiform hemorrhages will be observed much more frequently than formerly, in deaths from uræmic convulsions.

The other cerebral symptoms of uræmia, such as drowsiness, coma, and the typhoid state; *Mahomed* ascribes to œdema of brain-tissue, due to the same cause as that which produces the extravasations, viz., increased tension in the cerebral arteries and capillaries. The possibility of œdema of the brain, which has been denied by many eminent pathologists for mechanical reasons, our author regards as demonstrated, not only by the common occurrence of large hemorrhages, which could not take place if the pressure in the cerebral blood-vessels were not greater than the pressure outside them, but also by the fact that a lesion similar in nature to œdema does occur in acute Bright's disease, viz., the hyaline exudation in nervous tissue found by Sir *William Gull* and Dr. *Sutton*, the difference being merely that in the latter case the exudation is highly fibrinous and has coagulated so as to admit of being examined microscopically. Now, if the dropsy of Bright's disease be of mechanical origin, as is almost universally admitted, it is difficult to see why serum should not transude from the unusually delicate capillaries of the brain, as well as from those of other parts of the body, though perhaps not with the same facility, on account of the density of the surrounding structure and the intra-cranial pressure. If it be urged that uræmia occurs in chronic Bright's disease in which there may be no general dropsy, it may be replied that in this form of the affection the gradual increase in blood-tension is compensated by a gradual thickening of the capillaries, but that the time may come when this compensation is insufficient, and then, as in the supervention of an acute attack upon the chronic affection, either general dropsy or a partial one affecting the brain is liable to ensue. At all events, some of the slighter symptoms of uræmia, such as the characteristic headache of Bright's disease, may be readily explained by pressure upon the cerebral structures resulting from increased arterial tension, independently of exudution.

The two most serious objections to *Mahomed's* theory of the pathogenesis of uræmic convulsions are the absence of convulsive phenomena in certain diseases in which minute cerebral hemorrhages are not infrequently observed, and the fact that the similar convulsions of epilepsy are commonly supposed to be due to anæmia rather than hyperæmia of the brain. The first objection, namely that in purpura and allied conditions such hemorrhages are unattended by convulsions, is answered by the consideration that under these circumstances the hemorrhage is a *passive* exudation, and produces no increase of pressure upon or laceration of brain substance, whereas in Bright's disease the hemorrhages take place under *increased* blood-pressure, and therefore may more readily give rise to explosive evolutions of nerve-energy. As regards the analogy between uræmic and epileptic convulsions, *Mahomed* says: "However strong the evidence of anæmia of the brain in epilepsy may be, the side of hyperæmia is almost equally strong. If *Kussmaul* and *Tenner* think they have proved *experimentally* that anæmia of the brain produces convulsions, so also *Schroeder van der Kolk* has beyond doubt, I think, proved that dilatation of the capillaries occurs as a result of hyperæmia in epileptics, and this, be it remembered, not generally throughout the brain, which might be the result of general turgescence owing to impeded venous return, but in that centre in the medulla to which he refers the irritation producing the convulsions." *Hughlings Jackson's* theory of spasm of cerebral arteries as a cause of uræmic convulsions, based on the analogy between the convulsions of uræmia and those of epilepsy, must therefore stand or fall in great measure with the anæmic theory of epilepsy.

"Finally, it is interesting to note in this connection," says *Mahomed*, "that uræmic convulsions are more common in acute Bright's disease, while larger hemorrhages causing death from apoplexy occur more frequently in the chronic form and in more advanced life. It is not difficult to rupture the capillaries by increased blood-tension, owing to thinness of their walls, as seen in the kidney when bloody urine is produced, this being undoubtedly due to rupture of capillaries, while in chronic disease larger vessels have had time to undergo degeneration, so that they become equally, if not more liable to rupture."

The importance of still another factor in the pathogenesis of uræmia, viz., the diminished number and impaired respiratory capacity of the red blood-corpuscles has been recently insisted upon by Dr. *Paul Cuffer* in an interesting monograph on the blood-changes in uræmia (l. c.). These alterations in the blood are due, he holds, not so much to the urea, as to the poisonous and solvent action of carbonate of ammonia and kreatin. Experiments with all three of these substances showed that injections of carbonate of ammonia and kreatin into the blood of the lower animals were followed by a considerable reduction in the number of red corpuscles and a loss of their respiratory capacity, while no such results were obtained from urea. Thus the injection of urea in rabbits and dogs failed to modify the respiration, excite convulsions, or alter the number and appearance of the red corpuscles, whereas an injection of two drachms of carbonate of ammonia in dogs immediately produced vomiting, restlessness, and dyspnœa of the *Cheyne-Stokes* type. On examination of the blood, the red corpuscles were diminished by about twenty-five per cent in number, and were found to be less capable of absorbing oxygen. Injections of kreatin produced a similar, but less marked effect upon the red corpuscles, and merely lowered the frequency without altering the rhythm of the respiration. Experiments upon blood outside of the body gave similar results in the destruction of red corpuscles. Thus no alteration was noticed after adding urea, but carbonate of ammonia rapidly lessened the number of corpuscles, and kreatin produced the same effect, though to a less degree. The *clinical* results were of the same character. Loss of red blood-corpuscles and marked impairment in their respiratory capacity were noticed by *Cuffer* in his examinations of the blood in cases of uræmic coma and convulsions. That the losses of red corpuscles were not due to œdema or diarrhœa—when these symptoms were present—is rendered probable by *Brouardel's* observation that under these circumstances the blood becomes more consistent and relatively richer in formed elements.

The blood-changes above described, by producing a greater or less degree of asphyxiation of nerve-centres, explain many of the symptoms of uræmia, such as restlessness, convulsions, epileptiform attacks, and dyspnœa; the pathological condition being therefore analogous to that resulting from direct losses of blood in hemorrhage. Moreover, the intensity of the uræmic symptoms may vary in different cases according to the nature of the blood-poison, since experiment showed that carbonate of ammonia was far more destructive than kreatin in its action upon the red-corpuscles.

While thus insisting upon the importance of blood-changes as a factor in uræmia, *Cuffer* still admits the necessity of assuming the existence of *arterial spasm* in certain cases, in which marked uræmic symptoms were present without evidence of the blood-changes in question. This spastic contraction of arteries in uræmia *Cuffer* compares with the similar condition in lead poisoning, and suspects that it is due to a *sudden* retention of the urea, spasm being less likely to occur when the accumulation is gradual, and the tissues have become habituated to the presence of the poison.

The application which *Cuffer* makes of these views to the interpretation of *uræmic dyspnœa* is particularly interesting. Under this term are embraced only those forms of dyspnœa which are directly due to the uræmic poisoning, and are not traceable to pulmonary or cardiac lesions. *Cuffer* distinguishes two main varieties, one in which the respiratory rhythm is regular, and another in which it assumes the *Cheyne-Stokes* type. The first form often depends simply upon the blood-changes referred to, the respiration becoming hurried in order to compensate for the loss in the number and respiratory capacity of the red corpuscles, in the same manner as in the dyspnœa of leucocythæmia, chloranæmia, and the anæmia resulting from profuse hemorrhage. In illustration of the part played by paralysis of blood-cells, he cites a case of uræmic dyspnœa observed by *Brouardel*, in which no relief was afforded by inhalations of oxygen. In certain instances, however, the dyspnœa is immediataly due to spasm of the pulmonary arteries with consequent diminution of the respiratory surface.

Uræmic dyspnœa of the *Cheyne-Stoke's* type *Cuffer* subdivides into two varieties, one associated with restlessness and urgent respiration, and the other with a more quiet form of breathing. These two forms, he supposes, depend upon different blood-poisons, as experiments showed that the former could be induced in animals by injecting carbonate of ammonia, and the latter by injecting kreatin. *Cuffer's* explanation of the details of the phenomena differs from that generally given, as he assigns to the symptom a pulmonary rather than cerebral origin. The dyspnœa, he holds, starts in a spastic contraction of the pulmonary arteries, and not, as *Filehne* asserts in his recent work, in spasm of the arteries supplying the respiratory centre. The period of respiratory pause is due to the induction of a state of apnœa, in which breathing ceases, because the blood which has been hyperoxidized by the rapid action of the heart and the diminished arterial pressure in the lungs* no longer stimulates the respiratory centre. At the end of the respiratory pause the arterial spasm is renewed, and starts another cycle of respiratory phenomena. Although this form of uræmic dyspnœa is usually to be regarded as a fatal omen, because it occurs most frequently in an advanced stage of renal disease, it does not always possess this serious significance. Thus *Cuffer* saw it recur frequently for several years in a case of interstitial nephritis complicated by gout, and also at the outset of an attack of nephritis induced by exposure to cold.

With respect to the *temperature* in uræmia, two contributions have recently appeared which deserve a brief notice. *Strümpell*, in an article on the influence of uræmia on the temperature of the body (l. c.), presents the following conclusions based on an analysis of fifty cases of uræmia: Uræmia possesses no characteristic temperature curve. Both elevations and depressions are noticed in different cases. In mild forms of uræmia, these changes are slight, but in severe cases the temperature may rise as high as 41.5° (106.7° F.), especially when there are chills and sweats (without complications, pneumonia, etc.), or, on the other hand, may sink to the so-called collapse temperatures. The temperature in uræmia varies, therefore, in prognostic significance according as the temperature remains near the normal, or deviates from it by either a considerable elevation or a considerable depression.

In a still later article, *McBride* (l. c.) shows that the uræmic state is frequently accompanied by other conditions which, according to *Wunderlich*, *Hirtz*, and other authorities, directly tend to lower the body temperature, and that this fact may explain the depressed temperatures frequently noticed under these circumstances. At the same time he reports two cases of *elevated* temperature in uræmia and maintains (in opposition to the dictum of *Bourneville*)† that elevated temperatures are generally found in uræmia when these depressing conditions are absent. Among these depressing conditions *McBride* enumerates excessive vomiting or diarrhœa, dropsy, disorders of circulation from organic disease of the heart, old age, inanition, and the morbid states resulting from suppression of urine in obstruction of the ureters, or diseases of the bladder and urethra. It is probable, therefore, he concludes, that a low temperature will be observed:

"1. In cases of renal disease secondary to diseases of the urinary tract, especially when accompanied by complete suppression of urine.

---

* M. *Biot* (*Lyon Médical*, 1876; Nos. 50 and 51) found in a case of well-marked *Cheyne-Stokes* respiration in the Hôtel Dieu, at Lyons, that the cardiac pulsation were much more frequent during the period of apnœa than during that of dyspnœa, while the sphygmographic tracings showed that the arterial tension was lowered during the apnœa.

† *Bourneville* in his "Etudes cliniques et Thermométriques sur les Maladies du Système Nerveux; deuxième partie—Urémie et eclampsie puerpérale, 1872," holds that in uræmia low temperatures are observed, while in puerperal eclampsia the temperature is always elevated, and bases upon this supposed fact his theory as to the non-uræmic origin of puerperal convulsions.

"2. In uræmia occurring in the aged.

"3. In uræmia occurring in the course of very chronic renal disease, in which there may be vomiting, diarrhœa, or hemorrhages.

"4. In uræmia in the cancerous cachexia, and possibly in marasmic conditions."

In the chapter on *Urinary Casts* in *Ziemssen's* Cyclopædia (Vol. XV., pp. 86 and 87), *Bartels* takes the ground that urinary casts are formed by coagulation of albumen and its derivatives, and are therefore always associated with excretion of albumen in the urine. The association, he holds, is generally contemporaneous, but not infrequently casts are not discharged until some time after complete disappearance of the albuminuria to which they owe their origin. How long an interval may elapse between the formation and the discharge of casts *Bartels* does not attempt to decide. With this dictum as to the necessary dependence of casts upon albuminuria, Dr. *Finlayson* takes issue in an article "on the occurrence of renal tube-casts in non-albuminous urine" (l. c.). He reports a number of cases in which the presence of casts in the urine continued for variable periods, sometimes for several weeks after the last trace of albumen had disappeared; though these instances, it may be objected, may have been simply cases of prolonged retention. Other instances, however, are given, which apparently do not admit of such an explanation. Thus in the case of a healthy young man suddenly attacked with inflammation of the cæcum, the urine was repeatedly examined from the outset of the disease, through a period of nearly four months, without detection of albumen at any time, and yet hyaline tube-casts, generally only a few, but sometimes in large numbers, were found at each examination. The urine was usually of high specific gravity, loaded with urates and uric acid, and for several weeks contained sugar in greater or less amount. *Finlayson* offers no explanation of the production of casts in this case, further than to suggest that it may have been due to irritation of the urinary tubules by the loaded state of the urine. The glycosuria he excludes as a cause, for the reason that the tube-casts were most numerous while the urates and uric acid were most abundant, and continued to be found after the urine ceased to respond to Fehling's test. This conclusion is supported by other cases in which casts, both hyaline and epithelial, were found in connection with renal calculus and gravel, unaccompanied by the presence of albumen, as well as by the fact that tube-casts are almost invariably found in marked cases of jaundice, and, as a rule, without albuminuria. *Nothnagel* has observed that tube-casts are almost always present in deeply-jaundiced urine, while albumen is not found in more than one-third of the cases. This fact points to tubular irritation during the excretion of bile, and *Finlayson* suggests that the source of this irritation may possibly be found in the "brownish-black, angular granules" described by *Frerichs* in his work on "diseases of the liver," as present in the urine and filling the urinary tubules in cases of deep jaundice.

In an article "on some points in the minute anatomy of the kidney, and their relation to the pathological anatomy of tubular casts" (l. c.), Dr. *Southey* protests, upon anatomical grounds, against the view still very generally held, that the larger casts, and those which present most distinct cellular elements in their interior, are derived from the large tortuous tubules. It is certain, he says, that nothing larger than nuclei, blood-cells, or leucocytes can pass down from the tortuous or glandular part of the tubules through *Henle's* loops so as to reach the straight collecting-tubes. The channel of a down-looper is so narrow that it would be impossible for even a single one of the cubical columnar epithelial cells which line the glandular portion of the tortuous tubes to squeeze itself through in its ordinary shape, to say nothing of the still greater difficulty in transit when these large cells are massed together, to form a so-called desquamative cast. The only elements of casts which can fairly be referred to the upper portions of the tortuous tubes are mere derivatives and detritus in the form of finely-granular plasma containing fatty dottings, oil globules and nuclei, so that whatever the orginal source of casts may be, their *form* is always moulded in the excretory system of tubes. "When, therefore, a cast is assumed to be derived from the profounder tissue of the kidneys, and to have a relatively graver importance attached to it because cells are seen in its interior, an error is committed based on ignorance of the minute anatomy of the organ."

Southey admits three general varieties of casts: *granular* casts, which originate in degeneration of renal epithelium, the individual cells melting down into masses more or less homogeneous or granular, but preserving no outline of their original cell-forms; *waxy* casts, formed by "perverted or abnormal secretions derived from disordered functioning of the secreting-cells;" and *blood-plasma* casts, which consist of blood fibrin, entangling more or less blood elements. "The largest casts are most likely moulded in the large gathering mains near the papillary orifices; the medium-sized casts are moulded in the straight collecting tubes or ascending branches of the third order, while the smallest, finest, most wavy and hyaline, so-called fibrinous casts, are probably chiefly moulded in *Henle's* down-looping canals."

Besides these general forms of casts, *Southey* differentiates several subvarieties, which possess special clinical significance. Casts, in which are entangled distinctly formed cell elements, such as leucocytes, white or red blood-cells, point to a derivation from tortuous or excretory tubes, while hyaline cylinder casts, from their form and size, are probably formed in *Henle's* loops. Moreover, the more granular the cast, the longer it may be assumed to have resided in its matrix mould, and "the more dotted with fat globules casts are, the more certainly do they appoint permanent obstruction to the circulation through those portions of the kidney from which the colloids which form them are derived, and permanent damage, not to the tubes only, but to the capillaries and interstitial structures of the kidney."

"The *chemistry* of casts may be vaguely described as 'some colloid.' Two qualities of colloid may, however, for clinical purposes, be distinguished; the one derived from the upper glandular portions of the renal tubes, an abnormal secretion from its glandular cells, giving a yellow, waxy look, and highly refracting features to the cylinders formed of it; the other, a fibrino- or albumino-plastic colloid, essentially whiter and more transparent than the former, derived from the setting of the blood-plasma which has transuded into the tubuli from the blood-vessels at a lower part of their course."

The question as to the origin of fibrin cylinders has received an important contribution in a recent article by Dr. *Theodor Langhans* (l. c.), giving the results of his observations on the changes which the contents of uriniferous tubules undergo in nephritis preparatory to their transformation into casts. His conclusions accord very closely with those of *Axel Key*. According to *Langhans*, many fibrin cylinders are formed by fusion and disintegration of cells, which have escaped into the lumen of the urine canals. These cells may be either desquamated epithelium or lymph-corpuscles, or red blood-globules. The process of conversion into cylinders takes place in different ways. In most cases, the epithelial cells and lymph-corpuscles appear to break down into a finely granular mass, the origin of which may be indicated by the presence of individual cells, and especially nuclei. As the process advances, the latter also disappear by granular disintegration, and the lumen of the canal becomes filled with a more or less consistent, uniformly granular cylinder. The shining, hyaline appearance begins at the periphery, while the centre still remains granular for some time, and may even contain nuclei and cells, but later the same change takes place here also. Essentially the same stages of conversion are noticed in the red blood-corpuscles. These also disintegrate into a finely granular mass, in which the contours of individual cells can no longer be recognized, and finally the granular mass becomes homogeneous. Both the granular mass, however, and the resulting homogeneous cylinder are distinguished from similar forms originating in colorless cells by a distinctly yellow tinge, while the granules possess a more refracting property. In some instances, the transformation into a shining, homogeneous cylinder appears to take place *directly*, without a previous stage of granular disintegration.

Langhans admits also a second mode of origin for casts, although the evidence is less conclusive here than in the case of origin from epithelial disintegration. Many cylinders, he holds, particularly the homogeneous pale ones, may be regarded as a *secretion-product* of epithelial cells. Not infrequently the lumen of the uriniferous tubules, especially of the tubuli contorti, is found to be occupied by granular masses presenting different relations to the epithelium. Thus, in some instances, numerous pale granules are seen irregularly distributed in the lumen, lying rather towards its periphery, and attached to the inner surface of the epithelium, which, as a rule, is not smooth and sharply defined, but granular, while the granular protoplasm of the cells passes gradually into the loose granular mass filling the lumen. In other cases, the granules are grouped together in the form of reticula, which occupy the central portion of the lumen, and send out prolongations to the epithelium, so as to present stellar forms on transverse section. The central portions of these reticula are frequently broad, and of a homogeneous, bright appearance, resembling that of pale cylinders. Besides the stellate masses, others may be seen in the form of small spheres, or of crescents with a pale, granular, concave border. The spheres are optically identical with the hyaline albumen-drops, which are often observed exuding from living cells. *Langhans* concludes, from these appearances, that both the granular and the hyaline masses are derived from the epithelium by a process of granular desquamation, and that while the granules, if present in large numbers, coalesce to form hyaline reticula, crescents, etc., the *hyaline* spheres are generally discharged as such from the cells. To the objection that the appearances in question may be cadaverous, he replies that they are found in fresh bodies kept at a cool temperature, as well as in sections which have been quickly treated with hardening reagents.

According to both of the above theories, fibrin-cylinders consist essentially of metamorphosed cell-protoplasm. Whether there be a third mode of origin, viz., from exuded blood-fibrin, as *Weissgerber* and *Perls* (l. c.) claim to have demonstrated, *Langhans* does not venture to decide upon the basis of his own observations, but contents himself with saying that he has never seen any fibrin-cylinders which could not be accounted for on one or other of the theories supported by him.

ANASARCA.

BIBLIOGRAPHY.

Cohnheim, J., and Lichtheim, L.: Ueber Hydrämie und hydrämisches Oedem. Virchow's Arch., 1877, XCVI., 106.

In this important contribution to the study of the pathogenesis of œdema, *Cohnheim* and *Lichtheim* give the results of numerous experiments made upon the lower animals for the purpose of determining the relations of hydræmia to œdema. A brief abstract of this long article must suffice.

To determine the effect of dilution of the blood in producing œdema, a large number of experiments were made by injecting various solutions into the circulation of the lower animals. A six-per-cent solution of chloride of sodium was generally used, because, unlike the pure water employed by *Magendie* in his experiments, it does not dissolve the red blood-corpuscles. The introduction of the solution was continued until the death of the animal. This result occasionally occurred *suddenly*, with symptoms of overwhelming pulmonary œdema; but, in the great majority of cases, death took place *gradually*, with signs of imperfect decarbonization of the blood, feeble action of the heart, or convulsions, In all the cases, even when the largest injections were used, up to 92 per cent of the weight of the animals, *there was no evidence of cutaneous œdema,* the subcutaneous connective tissue being invariably *perfectly dry.*

Serous transudation, to be sure, was found, but always in situations which are rarely the earliest or chief localization of so-called hydræmic œdema. Thus copious transudations of water occurred from the glands, and into certain lymphatic channels and tissues. The secretions of the kidneys, salivary and buccal glands, gastric and intestinal glands, the liver, and the lachrymal glands were profusely increased. The rapidity of the circulation in the thoracic duct was, moreover, notably increased; in the cervical lymphatics the circulation was also more rapid than usual, though much less so than in the thoracic duct, while no change was observed in the lymphatics of the skin and muscles. More or less ascitic fluid was always found. The mucous membrane and submucosa of the intestines and stomach were œdematous to a marked degree, as were also the pancreas, and, to a less extent, the kidneys. On the other hand, the pericardial and pleuritic sacs contained no fluid, nor was there any œdema of the lungs, except in the few instances where death resulted from this accident. The central nervous system and subcutaneous connective tissue were also invariably free from œdema.

The most striking results obtained were, therefore, the dropsical effusion into the abdominal cavity, and the œdematous swelling of the pancreas, and gastro-intestinal wall. In order to determine whether the œdema in these situations might be due to venous stasis in the portal radicles, from obstruction to the venous blood in passing through a second capillary system in the liver, other experiments were made, in which the portal vein was connected with the external jugular or the femoral vein, so as to allow the portal blood to pass directly to the heart. No difference was noticed, however, from this modification of the operation, and *Cohnheim* concludes, therefore, that the explanation of this peculiar localization of œdema is probably connected with the normal function of these parts in excreting water from the blood.

Still further experiments were instituted for the purpose of ascertaining whether the œdema was produced by dilution of the blood (hydræmia), or by increase of the contents of the vessels (hydræmic plethora), or by the salt contained in the injected fluid. Infusions were tried with a three-per-cent solution of grape sugar, other saline solutions, a dilute solution of albumen, diluted blood-serum, and undiluted dog's blood, but in all cases essentially the same results were obtained. On the other hand, the artificial induction of simple hydræmia in dogs by daily abstractions of blood, and replacing the losses with equal amounts of chloride of sodium solution, was unattended by any œdema in the organs found water-soaked in hydræmic plethora. The conclusion seems inevitable, therefore, that, in the animals experimented upon, the determining factor in the production of œdema was not the *dilution* of the blood or the increase of the *relative* amount of water, but rather the hydræmic *plethora,* that is, the increase of the *absolute* amount of water.

The application of these results to the explanation of œdema in renal disease, where the chief localization of serous transudation is in the subcutaneous connective tissue, is at first sight not very apparant, but *Cohnheim* presents several considerations which throw some light upon the matter. In the first place, we have seen that the increased serous transudation, when the blood contains a large excess of water, takes place by preference from quite definite vascular districts, while in the majority of all the vessels the amount of escaping fluid remains the same, however large the quantity of injected water. The latter statement is true, however, only of vessels which perform their function normally; indeed, there is much evidence that in morbid conditions of the vascular membrane it is by no means indifferent whether the circulating blood be of normal concentration or of an unusually watery quality. If the vessel-lesion be insufficient of itself to produce œdema, this result ensues as soon as either simple hydræmia or hydræmic plethora is established by the means previously referred to. If a slight œdema already exist, as in the case of wounds inflicted some time before the experiment, a marked increase of the œdema will appear under the eye of the observer during the course of the infusion. Similar results may be obtained also with chloride of sodium injections after production of morbid conditions of the cutaneous vessels by painting the skin with tincture of iodine, or by exposing the shaved skin of a dog to the direct rays of the sun for one or two hours.

In view of these facts the thought naturally suggests itself, whether the direct cause in so-called hydræmic œdema in the human subject should not be sought in morbid conditions of the walls of the vessels, particularly those of the skin. The intervention of this factor would enable us to understand why no œdema may occur even in the presence of a high degree of hydræmia or hydræmic plethora, while at the same time the undeniable relations between these conditions and œdema are thereby readily explained. For although the occurrence of œdema may be immediately determined by vessel-changes, the latter may be in themselves insufficient to produce this result without the co-operating influence of hydræmia or hydræmic plethora; at least it was always found, when vessel-changes were artificially established, that the amount of œdema corresponded with the amount of hydræmic plethora.

It is not to be supposed, however, that in *ambulant* œdema there is necessarily a corresponding fluctuation in the functional derangement of the vessel-walls. Slight venous stasis, determined by the force of gravity, must also be admitted as a co-operating influence, as shown by the more frequent localization of hydræmic œdema in the more dependent parts of the body, and the same may be said of the local congestions excited by the physiological play of vaso-motor nerves. Congestive hyperæmia, by itself, never produces œdema, but when combined with influences which impair the vessel-walls, certainly favors the occurrence and increase of serous transudation from the vessels.

The relation between changes in the cutaneous vessels and affections of the renal parenchyma is clearly shown by the occurrence of the latter in connection with various forms of inflammatory changes in the skin. In scarlet fever, for instance, œdema is rarely present in appreciable amount, notwithstanding the inflammatory changes in the cutaneous vessels, until the discharge of urine is diminished by the renal complication, and hydræmic plethora is established. So also nephritis frequently results from chilling of the skin, while albuminuria is well known to be a common occurrence in extensive burns, and may be artificially excited by scalding or varnishing the skin of the lower animals.

As regards the œdema noticed in phthisis, cancer, and other cachectic conditions independently of any renal complication, it is probable that simple hydræmia, if long continued, may of itself injure the walls of the cutaneous vessels so as to permit copious serous transudation. The normal function of the vessel-wall depends upon the maintenance of the normal circulation within the vessels. Even a short interruption may injure the vessel-wall severely. Not only the oxygen of the blood, but probably also the presence of other constituents are essential to the performance of the normal function of the walls of the vessels, so that a high degree of impoverishment of the blood in solid substances may lead first to morbid changes in the structure of the vessels, and thus secondarily to the development of œdema.

CHRONIC BRIGHT'S DISEASE.

BIBLIOGRAPHY.

JOHNSON, GEO.: Granular Degeneration of the Kidney with Special Reference to the Theory of Arterio-Capillary Fibrosis. British Med. Journ., 1875, I., 444.—On the Muscular Arterioles, their Structure and Function in Health and in certain Morbid States.—IDEM: 1877, I., 443, 503, 539, 577.—On the Minute Anatomy of the Small Red Granular Kidney.—IDEM: 1878, I., 746.—FRÄNKEL, A.: Zur Pathologie der Nieren. Berl. klin. Wochenschr., 1875, No. 43-44.—GOWERS, W. R.: The State of the Arteries in Bright's Disease. British Med. Journ., 1876, II., 743.—THOMA, R.: Zur Kenntniss der Circulationsstörung in den Nieren bei chronischer interstitieller Nephritis. Virchow's Arch., 1877, LXXI., 42, 227.—EWALD, C. A.: Ueber die Veränderungen kleiner Gefässe bei Morbus Brightii und die darauf bezüglichen Theorien. Virchow's Arch., 1877, LXXI., 453.—MAHOMED, F. A.: Arterio-Capillary Fibrosis and its Clinical Recognition, Lancet, 1877, II., 232.—On Chronic Bright's Disease and its Essential Symptoms.—IDEM: 1879, I., 46, 76, 149, 261, 399, 437.—Some of the Clinical Aspects of Chronic Bright's Disease. Guy's Hosp. Reports, 1879, 363.—ALLBUTT, T. C.: Pathology of Granular Kidney. British and Foreign Med. Chir. Rev., 1877, CXX., 279.—On Mental Anxiety as a Cause of Granular Kidney. British Med. Journ., 1877, I., 157.—SENATOR, H.: Beiträge zur Pathologie der Nieren. 1. Ueber chronische interstitielle Nephritis. 2. Ueber die Beziehungen der Herzhypertrophie zu Nierenleiden. Virchow's Arch., 1878, LXXIII., 1, 313.—DICKINSON, W. H.: Diseases of the Kidney and Urinary Derangements. Part 2, Albuminuria, 1878, 536.—Croonian Lectures on the Pathology and Relations of Albuminuria. British Med. Journ., 1876, April

15th, et seq.—GRAWITZ, P., and ISRAEL, O.: Experimentelle Untersuchung über den Zusammenhang zwischen Nierenerkrankung und Herzhypertrophie. Virchow's Archiv, 1879, LXXVII., 315.—WEIGERT, C.: Die Bright'sche Nierenerkrankung vom pathologisch-anatomischen Standpunkte. Samml. klin. Vorträge, Leipzig, 1879.—COENIL, V.: Nouvelles observations histologiques sur l'état des cellules du rein dans l'albuminurie due à la néphrite parenchymateuse et la néphrite interstitielle. Jour. de l'anat. et de la physiol., 1879, XV., 402.—ROSENSTEIN, S.: Ueber Morbus Brightii und primäre Nierenschrumpfung. Wien. med. Presse, 1879, XX., 1088.—SAUNDBY, R.: The Polyuria of Granular Kidney. The Birmingham Med. Review, 1878, April.—Relation between Kidney Disease and Hypertrophy of the Heart.—IDEM: Jan., 1880.—The Histology of Granular Kidney. Med. Times and Gaz., 1880, 1, 398.—The Diagnosis of Granular Kidney. The Birmingham Med. Rev., July, 1880, 193.—DA COSTA, J. M., and LONGSTRETH, M.: Researches on the State of the Ganglionic Centres in Bright's Disease. Amer. Journ. of Med. Sc., July, 1880.

The vascular lesions of chronic interstitial nephritis have recently been studied by Dr. *Richard Thoma*, of Heidelberg (l. c.), with special reference to the disturbances of the renal circulation in this affection. Numerous experiments were first made with both normal and granular kidneys to ascertain their comparative permeability to injections through the renal artery. Fresh kidneys were used, which had been removed at the earliest practicable moment (generally from six to twelve hours after death), in order to obviate so far as possible the disturbing influence of rigor mortis of the muscular layer of the renal vessels. Human blood not being obtainable in sufficient quantities, defibrinated ox-blood was used in a few instances, but generally *Thoma* employed, as more satisfactory in their results, either a solution containing 8 per cent of gelatin and 7.5 per cent of chloride of sodium, or an aqueous solution of gelatin without the salt. These comparative experiments showed not only that the outflow from the renal vein was *absolutely* less in granular than in normal kidneys, but also that the outflow as compared with the amount injected into the renal artery was *relatively* diminished in granular conditions of the organ. Two causes are assigned by *Thoma* for this result: 1, the reduced calibre of the renal artery and its branches, and the obliteration of numerous minute vessels by the interstitial process, and 2, the increased permeability of the vascular walls. The latter factor retards the circulation by permitting the more rapid escape of fluid from the vessels into the surrounding tissue, and thus disturbing the peripheral layers of the blood-current, while on the other hand the blood-vessels themselves are directly compressed by the transuded fluid. It was noticed, however, that the diminution in the outflow of injections did not always stand in direct proportion to the amount of renal disease. Thus, while in less marked forms of contracted kidney the resistance to the blood-current generally corresponded to the degree of interstitial proliferation, quite a free transit of injections was sometimes observed in advanced stages of the affection, probably on account of certain changes in arterial distribution to be more fully described later.

Careful comparative measurements were also made, upon healthy and contracted kidneys, of the sectional areas of the renal artery, of the interlobular artery midway between the pyramids and capsule and also near the capsule, of the vas afferens glomeruli and of the Malpighian vessels. These measurements were made upon healthy kidneys, from the time of birth up to 43 years of age, and were compared with the changes in size of the organ at corresponding periods of life. It was found, from an average of 60 measurements and weighings, that the normal kidney grows between birth and the 36th year from a weight of 14 grammes to one of 150 grammes, while the sectional area of the renal artery increases from 2.5 to 36 square millimetres, thus showing that the calibre of this artery increases much more rapidly than the size of the kidney, and, therefore, sends to the organ a much greater supply of blood relatively to the size of the kidney, after the 36th year than in early life. *Thoma* attaches importance to this relatively increased vascularity after the completed growth of kidney as favoring the influence of direct causes in the production of connective tissue proliferations within the kidney in middle and advanced life. Measurements of the interlobular artery and smaller arteries gave a similar result. In *contracted* as compared with healthy kidneys, the sectional area of the renal artery and smaller vessels was generally slightly diminished, but sometimes appeared increased, owing partly to structural changes, such as connective tissue thickening, calcification, and the presence of atheromatous masses, and partly also to the prevention of elastic contraction and collapse by the changes referred to. The general result showed that the renal arteries, large and small, are considerably increased in size relatively to the weight of the kidney.

The effect of this increased arterial flux to contracted kidneys and retarded current in the vessels is naturally to raise the arterial pressure, and *Thoma* accordingly found, by introducing a mercurial manometer into the walls of the renal artery, that, whereas in the healthy organ the lateral pressure of injections averaged 14.5 cm. of mercury, in granular kidneys it rose to 16 cm. In the smaller vessels the difference must be considerably greater.

In the second portion of his article, *Thoma* considers at greater length the pathological increase in permeability of the walls of the vessels. Experiments were made with a solution containing gelatine, Prussian blue, and chloride of sodium, and in some instances cinnabar—with the result of demonstrating a marked difference in the amount of transuded coloring matter in granular as compared with sound kidneys. The principal sites of transudation were the Malpighian capsules and the tubuli contorti. A general correspondence was observed between the frequency of transudation and the amount of connective tissue proliferation and contraction, but the colored masses were noticeably much more numerous in the still intact portions of the cortex, probably because the arteries were here less distorted and compressed by the contracting connective tissue, and therefore admitted a more forcible current. The increased permeability did not seem to be caused by the hyaline or fibrous degeneration of minute arteries described by *Johnson* and *Gull* and *Sutton*, as it was the apparently unchanged glomeruli of the less diseased renal lobules which especially favored these transudations.

Similar transudations, though in a much less marked degree, were found also in the district of the capillary tract. Here blue masses were often observed lying in the fissures of the connective tissue, especially in the granulating and softer tissues which characterize the beginning of the morbid process, rendering it probable that this increased permeability of the capillary wall stands in intimate causal connection with the development of the neoplastic process.

Although the morbid changes described directly tend to obstruct the blood-current through the kidney, there are others, in the form of modifications of the arterial distribution, which probably facilitate the circulation in contracted kidney. The most frequent of these modifications is the establishment of a direct communication between the vas afferens and the vas efferens in numerous glomeruli. Such an anastomosis may be effected by gradual obliteration of the vessels of the tuft in two ways: either the vascular loops may themselves undergo a connective tissue transformation, corresponding with the so-called hyaline degeneration, or *fibrous endarteritis*, as *Thoma* prefers to call it, resulting in the obliteration of one loop after another, until finally the single loop remaining is cut off from the stem of the glomerulus, and the vas afferens enters into direct communication with the vas efferens; or the same result may be produced without structural changes in the vessels through compression of the tuft by connective tissue proliferation starting in the capsule, and gradually filling the capsular space. In marked cases, the two forms of atrophy may be readily distinguished. In the first, the outlines of the thickened glomerulus may be seen in the midst of the hyaline mass, while in the other form the vascular tuft cannot be recognized, and the capsular space is filled with concentric layers of neoplastic tissue.

Still another alteration of arterial distribution is described by *Thoma*, in connection with cystic transformation of the glomeruli. Here also a direct communication is established between the vas afferens and the vas efferens; but, in addition to this change, the vas efferens and its branches undergo a peculiar transformation, resulting in the development of a broad capillary network with numerous anastomoses on the surface of the cyst, connecting also with the vas afferens, so that the blood passes from comparatively large arterial branches into a system of wide-meshed capillary vessels, and then empties directly into venous radicles. Furthermore, as *Ludwig* has shown, a few of the vasa afferentia in normal kidneys give off branches which open directly into the capillary tract. In the granular kidney, these branches are dilated and new ones formed. A further derivative influence is exerted by collateral anastomoses with neighboring arteries, the supra-renal, phrenic, lumbar, and their minute branches. In abnormally high pressure within the renal vessels, these anastomoses may carry off a much larger portion of blood than usual.

As regards the structural alteration in the walls of the vessels, *Thoma* regards the change as identical with the endarterial process, described by

Köster, *Friedländer*, *Trompetter*, and others, as frequently occurring in connection with new formations of connective tissue. A glistening mass, resembling amyloid substance, but giving no corresponding reaction, lies on the outer surface of the endothelial layer of the vessel, being bounded externally by the basement membrane, or, when this is wanting, by the circular muscular layer. The lumen of the vessel is frequently, but by no means always, irregularly bulging, being narrower and broader by turns, but there is no such general and considerable narrowing as *Gull* and *Sutton* suppose. Their mistake upon this point seems to have arisen from a faulty mode of measurement, which merely showed that the vascular wall was thickened as compared with the lumen. If the smaller arterial branches, for example the middle portion of the interlobular artery, or the vas afferens, be carefully measured in both healthy and contracted kidneys, it will be found that, notwithstanding the very considerable thickening of the wall, the absolute size of the lumen is generally unchanged, or is even increased. In some places, however, where there is much thickening of the wall, the entire vessel may be considerably narrowed and contorted in shape. The muscularis is apparently stretched by the neoplasm, and at certain points is decidedly thinned or entirely wanting, its cellular elements being atrophied and replaced by the thickened connective tissue of the adventitia, which passes over without sharp limits into the products of proliferated interstitial tissue.

In the larger branches of the renal artery, the glistening masses situated between the endothelial layer of the intima and the elastic lamella consist of closely apposed longitudinal bundles of fibres with numerous spindle-shaped cells. In marked cases, these bundles are more distinct, and present rhomboidal interspaces with oval nuclei surrounded by a delicate granulation, indicating a thin protoplasmic body. Exceptionally, the thickening of the vessels at some points is much more voluminous, so that the lumen is narrowed by several millimetres, or may be completely occluded. In such cases the tissue intervening between the endothelium and the elastica contains numerous capillaries, and large round or oval nuclei, surrounded by a broad layer of protoplasm. The interspaces of this meshwork are occupied by protoplasmic bodies, containing large nuclei, the whole appearance indicating a rapid growth of new connective tissue. Analogous changes are found in the renal artery, only here the connective tissue process in the tunica media is complicated by calcification and softening atheromatous masses.

It is clear from *Thoma's* description of the arterial changes observed by him in chronic interstitial nephritis that he has failed to discover the muscular hypertrophy upon which so much stress is laid by Dr. *Geo. Johnson* and his followers, and that his (*Thoma's*) views coincide very closely with those advanced by Sir *Wm. Gull* and Dr. *Sutton*, so far at least as the renal process is concerned. No mention is made of any hypertrophy of the arterial muscular coat; in fact, the changes in this tissue, when any were noticed, were rather in the direction of atrophy from stretching and compression by the proliferated connective tissue.

The relations of hypertrophy of the heart to diseases of the kidney have recently been fully discussed by *Senator*, of Berlin, in *Virchow's Archiv* (l. c.), with special reference to the form of cardiac hypertrophy, commonly associated with chronic interstitial nephritis. In agreement with *Roberts, Dickinson, Grainger-Stewart*, and most English writers, with *Kelsch* and *Lecorché* among French authorities, and with *Bartels* in his opposition to the prevalent German opinion, *Senator* regards cirrhosis of the kidney in its typical form as a distinct affection, which is not necessarily associated with or dependent upon parenchymatous or diffuse nephritis, and which is characterized, at least in its initial and middle stages, by the secretion of *excessive* quantities of pale, clear urine of *low* specific gravity, with either moderate amounts of albumen or none at all, and generally containing, except towards the end of the disease, a *normal* quantity of urea and other specific ingredients of the urine. Furthermore, this affection is accompanied, in a large proportion of cases, by hypertrophy of the left ventricle of the heart. The occurrence of this cardiac lesion in *all* forms of Bright's disease, but especially in *renal cirrhosis*, has been recognized ever since the time of Bright, but the existence of two forms of cardiac hypertrophy in this connection, viz., *eccentric hypertrophy or hypertrophy with dilatation*, and *simple* or *concentric* hypertrophy, and the important bearing of this fact upon the pathogenetic relations of cardiac hypertrophy to renal disease, have not attracted sufficient attention.

In pure forms of chronic interstitial nephritis, the cardiac hypertrophy is generally of the simple variety. In proof of this proposition, *Senator* adduces his own experience as well as the evidence afforded by the statistical tables of *Traube, Dickinson, Bartels, Buhl*, and *Galabin*. On the other hand, in chronic parenchymatous nephritis, as well as in renal cirrhosis when the latter is complicated by arterial sclerosis and roughness of the aortic valves, or by inflammation of the endocardium, pericardium, or pleura, the cardiac hypertrophy is generally accompanied by dilatation.

That the heart should become hypertrophied when conditions such as these are present to obstruct the outflow of blood from the heart is readily intelligible, but the explanation is less easy when no equivalent mechanical obstacle can be demonstrated.

To consider first the cardiac hypertrophy associated with *chronic parenchymatous nephritis*—and here hypertrophy of the heart is admitted to be much less frequent than in renal cirrhosis—it will be seen that, after deducting the cases in which valvular defects or other obstructive lesions are present, only isolated cases remain to be otherwise explained. That some obstacle to the outflow of blood from the heart exists also even in these cases is clear from the fact that the hypertrophy is associated with *dilatation*, and such obstruction, in the absence of gross mechanical interference, can be found only in increased aortic tension. Now in chronic parenchymatous nephritis there are no general arterial changes, such as exist in renal cirrhosis, by which the blood-pressure in the aorta can be raised, nor can the retention of water in the blood, in consequence of deficient urinary secretion, produce this result permanently, for an excess of water is soon discharged from the blood, either by increased activity of vicarious secreting organs or by the occurrence of œdema. We are compelled, therefore, to refer the abnormal aortic pressure to blood-changes, especially the overloading of the blood with urea. *Ustimowitch* and *Grützner* have both shown that urea injected into the blood increases the aortic pressure, so that we have here a sufficient explanation for certain cases of cardiac hypertrophy, but probably only for a small minority of instances. For, to produce this result, not only must the accumulation of urea in the blood be long continued or frequently repeated, but the general nutrition must at the same time be well maintained, otherwise but little urea is formed, and atrophy rather than hypertrophy of the heart ensues. How rarely such a combination is met with in chronic parenchymatous nephritis need not be insisted upon.

In passing now to the relation of cardiac hypertrophy to *renal cirrhosis*, we encounter still greater difficulties. All theories hitherto advanced, except that propounded by *Gull* and *Sutton*, are based upon the supposition that the hypertrophy of the heart is secondary to the renal disease, and differ only as to the mode in which this result is produced, whereas there is no conclusive evidence that the hypertrophy of the heart may not be primary, or at least be a co-ordinate symptom of a general systemic disease. As the cardiac hypertrophy is here usually of the simple variety, that is, unattended by dilatation, the physical signs usually relied upon for the detection of ordinary forms of hypertrophy, such as depression of the apex beat in the longitudinal axis of the heart and increased dulness in the same direction, are here unavailable. So also the diagnostic indications for simple hypertrophy suggested by *Traube*, viz., abnormal resistance of the apex beat in its usual situation, a louder diastolic aortic sound of an elevated pitch and ringing quality, and a more forcible radial pulse with a normal condition of the arterial wall—these too are untrustworthy, because they are merely exaggerated normal conditions, and may be variously interpreted by different observers. It is quite possible, therefore, that simple hypertrophy of the heart may be present at the very outset of a chronic interstitial nephritis, and for a considerable time during its course, without being demonstrable by physical examination. We have consequently no means of determining the relation of events, or of proving a direct causal connection between the two lesions.

In purely interstitial disease of the kidney, there are no *renal* conditions which can explain the development of hypertrophy of the heart. The destruction of numerous blood-vessels in the kidney, or their compression by the interstitial exudation, is certainly insufficient to raise the blood-pressure in the aorta, for *Ludwig* has shown (*Virchow's Archiv*, Bd. LXXI., p. 42) that even the ligation of both renal arteries fails to increase the aortic tension, except for a very short time. Moreover, the copious secretion of urine in this affection naturally prevents the occurrence of increased arterial pressure from retention of an excess of water in the blood, even if we admit that such an accumulation could produce this result. Nor can retention of urea and other urinary ingredients in the blood be adduced as a cause of increased aortic pressure in this connection, because in renal cirrhosis, except towards the end of the disease, the excretory function of the kidney is well performed. Finally it remains to be explained why the cardiac hypertrophy in chronic interstitial nephritis is so frequently of the *simple* variety, and not eccentric as it is in the parenchymatous form of disease.

The possibility must, however, be conceded that, in these cases of apparently simple hypertrophy, a certain amount of dilatation may really be present, although disguised by the preponderance of muscular hypertrophy, or by excessive cadaveric contraction of the ventricle. *Bamberger* has shown that, in stenosis of the aortic orifice, the dilatation of the left ventricle may be so slight, as compared with the marked hypertrophy, that the cavity appears no larger than usual or even contracted. On careful examination, however, the septum will often be found to bulge towards the right ventricle, thus affording additional space for the left chamber of the heart. Whether such a condition obtains in the cardiac hypertrophy associated with renal cirrhosis *Senator* is unable to decide, but if this should prove to be the case, some obstacle to the cardiac circulation must be inferred here also. This can only be found in the increased aortic pressure, which is generally admitted to be present in renal cirrhosis, while the obstruction to the arterial circulation finds a satisfactory explanation in the more or less widespread thickening of the minute vessels (arteries and capillaries), which is perhaps the most striking lesion of this form of renal disease, and this too, whether the adventitia or the muscularis of the arteries, or both, are affected. So far the explanation rests upon the basis of well ascertained facts, but it is necessary first to decide upon the nature of these vascular changes, before any conclusion can be drawn as to their cause and their relation to renal cirrhosis on the one hand, and on the other to the cardiac hypertrophy which frequently accompanies it. For reasons already given, all theories which take the renal affection as the starting-point are inapplicable here, and the only hypotheses worth considering are the two advocated by *Gull* and *Sutton*, and by *Geo. Johnson* respectively.

Gull and *Sutton*, as is well known, hold that the morbid process in the vascular system—to which they have given the name "arterio-capillary fibrosis"—is the primary disease, in which the kidneys frequently, but by no means necessarily, take part. Now the fact that renal cirrhosis is accompanied by a marked tendency to connective tissue proliferations throughout the body, as shown by the frequent co-existence of cirrhosis of the liver, thickening of the splenic capsule, pleuritic adhesions, thickening and adhesions of the membranes of the brain and spinal cord, etc.; this fact points strongly in favor of *Gull* and *Sutton's* view, as there is no apparent way in which the systemic process can arise as the *result* of the renal disease. Furthermore, this theory, according to which the general arterio-capillary fibrosis may precede, accompany, or follow the renal affection, affords a satisfactory explanation of certain symptoms, which are often present at the very outset of the renal disease, or even before its first manifestations, and are very difficult to account for except on this hypothesis, such as the frequent severe headaches and the so-called "rheumatic" pains in the limbs and back.

According to *Johnson*, on the other hand, the starting-point of the disease is a defective constitution of the blood, such as obtains in gout, in the dyscrasia of inebriety, in chronic lead-poisoning, etc. This malnutrition of the blood produces degeneration and destruction of the renal epithelium, whose office it is to remove blood impurities, and the kidney consequently shrinks from loss of the epithelium and collapse of numerous tubules. As the intact portion of renal substance requires less blood, the supply is regulated by a corresponding contraction of the minute renal arteries, resulting, in time, in hypertrophic development of their muscular coat. In the same way, the muscular walls of other arteries of the body become hypertrophied, in consequence partly of the original altered composition of the blood, and partly of the retention of urinary excreta, until finally the heart also undergoes hypertrophy, as a result of the antagonism of forces. Aside from the forced explanation which *Johnson* gives for the muscular hypertrophy of the renal arteries, and from the mode in which he accounts for the contraction of the kidneys, his theory is justifiable in so far as it attributes the renal disease to dyscrasic conditions of the blood, which experience shows tend to induce cirrhosis of the kidney. The hypothesis is, however, open to the objection of multiplying causes where a single one is sufficient, since in renal cirrhosis there is usually no retention of urinary excreta, while in chronic parenchymatous disease, where such retention is present, the arterial changes in question are not observed, as *Ewald* has recently demonstrated. *Gull* and *Sutton's* theory, on the other hand, is entirely compatible in all its details with the supposition of a primary defect in blood composition.

If the disguised ventricular dilatation, which we have been considering, be not actually present, however, that is, if the cardiac hypertrophy associated with pure renal cirrhosis be truly of the simple variety, we have here to deal with a *primary* or *idiopathic* hypertrophy, which, as in the case of every hollow muscle, cannot be caused by obstruction to the evacuation of its contents, but must depend upon some condition which, by increasing the functional activity of the organ, disposes to abnormally strong and frequent contractions. Such a condition must be found in the dyscrasic composition of the blood, to which *Johnson* attributes renal cirrhosis, for a purely nervous origin of the hypertrophy, as in *Basedow's* disease, is probably out of the question. On this hypothesis, the admitted increase of arterial pressure can be neither the cause nor the effect of the cardiac hypertrophy. It is not the cause, otherwise we should have dilatation accompanying the hypertrophy; it cannot be the effect, for simple hypertrophy merely produces a quicker distention of the arterial wall, with a steeper line of ascent in the pulse tracing, without changing the average pressure in the arteries. This being the case, the increased arterial tension can be due only to the diffuse morbid process in the arterial system, which, as we have seen, directly heightens the pressure of its contents.

As regards the relation between the thickening of the arterial vessels and primary cardiac hypertrophy, this seems to be twofold. Either the *muscular elements* of the arteries may undergo hypertrophy in consequence of the overwork to which they are subjected by the force of the hypertrophied ventricle, as *Johnson* supposes, or the excessive tension of the arterial walls may set up chronic inflammatory changes of the kind reported by *Gull* and *Sutton*. Indeed, there is nothing to prevent our referring both forms of arterial change to abnormally powerful cardiac contractions. That the renal arteries should share to such an apparently disproportionate degree in the results of this increased arterial pressure may be accounted for by the special arrangement and abundance of the arterial vessels in the kidneys.

We may conclude, therefore, that, in all cases of cardiac hypertrophy associated with renal cirrhosis—whether the enlargement is only apparently or is actually simple—*thickening of the smaller systemic arteries is the essential factor in the production of heightened aortic pressure*, the difference in the two cases being that, in the one, the increased arterial tension precedes the cardiac hypertrophy as its *cause*, and, in the other, follows it as its *result*, or as a co-ordinate effect with it of a third factor (abnormal composition of the blood). Finally, in the present position of our knowledge, renal disease cannot be regarded as a *cause* of heightened blood-pressure and of changes in the organs of circulation, but only as a *result* or a concomitant manifestation of the same.

In connection with the present subject should be mentioned some recent experiments made by *Grawitz* and *Israel* (l. c.) to ascertain the effect of loss of secreting renal parenchyma in producing hypertrophy of the heart. Two series of experiments were made upon rabbits: in the first, the circulation in *one* kidney was arrested for from one and a half to two hours, by closing the renal artery on that side; in the second series, one of the kidneys was extirpated. As a result of the first procedure, acute parenchymatous nephritis was rapidly developed, followed in most cases, as early as the twelfth day, by changes which closely resembled, in their gross and microscopical appearances, granular atrophy of the kidney. In other cases, when bronchial catarrh, or pneumonia, or suppuration from the wound supervened, or when putrid fluids, even after boiling, were injected into the blood in non-toxic doses, it was noticed that, after the lapse of from fourteen to twenty days, there was no decrease, but rather an increase in the size of the organ. The renal epithelium, instead of being removed, as in the other cases, retained its position and presented marked fatty changes; in a word, the patho-anatomical picture was that of chronic parenchymatous nephritis.

The ultimate effect of this loss of renal parenchyma, whether produced by extirpation of one kidney or by the induction of chronic interstitial or parenchymatous nephritis, was strikingly different according as the animals were *young* or had attained *mature* growth. In the first group, the intact kidney rapidly increased in size until it reached a dimension equal to that of both kidneys in equally large rabbits. The growth of the animals was not arrested, and there was every reason to suppose from their liveliness and apparent good health that the intact kidney was performing the function of both organs, and that complete compensation had taken place. In the second group, consisting of *mature* animals, the compensation was less perfect. Here also the operations were immediately followed by an increase in size of the intact kidney, but the hypertrophy after the lapse of two or three months was found to be much less than in the first group. In some of these animals, death resulted from acute or chronic uræmia in its ordinary forms ; in others a state of imperfect compensation could be predicted during life from the impoverished nutrition and delicate health of the animals. Still other animals remained in good health for weeks and months, and in these the autopsy revealed a hypertrophic development of the left ventricle of the heart, equal in amount to the loss of renal substance. It was concluded, therefore, that after the loss of secreting renal parenchyma, whether induced by a cicatricial or fatty process in the kidney or by extirpation of one organ, a compensatory hyperplasia takes place in the other (sound) kidney, and that in young animals the function is thus completely resumed, so that no cardiac hypertrophy is noticed. In adult animals, on the other hand, when the hyperplasia of the intact kidney fails to replace the loss of renal substance, hypertrophy of the left ventricle may ensue, but the compensation can be complete only when the cardiac hyperplasia is equal to the lost weight of renal parenchyma.

Experiments were also made with the manometer introduced into one of the carotid arteries, for the purpose of ascertaining whether the cardiac hypertrophy could be due to increased arterial pressure. In no instance was such heightened pressure detected, even when marked hypertrophy of the left ventricle was found at the autopsy in connection with advanced granular or chronic parenchymatous lesions in the kidney. It is probable, therefore, our authors conclude, that, although the cardiac hypertrophy is doubtless dependent upon the diminished renal secretion, this result is not produced through the intervention of heightened arterial pressure, but rather by the direct action of retained urinary excreta, which stimulate the heart to overactivity, in the same manner as they stimulate primarily the intact kidney itself.

The pathological changes affecting the vascular apparatus in Bright's disease have recently been carefully studied in a series of sixty-two cases by Dr. *C. A. Ewald,* of Berlin (l. c.), who reports his observations in what may be regarded as a very important contribution to the *Gull-Sutton* and *Johnson* controversy. *Ewald* selected for microscopical examination the vessels of the pia mater covering the pons, not only because these vessels afford the most satisfactory preparations, and have been thoroughly studied in connection with other morbid processes, but also because the alterations here found may be regarded as typical of the general vascular changes in Bright's disease. In agreement with Dr. *George Johnson*, *Ewald* regards the morbid appearances described by *Gull* and *Sutton* under the term "arterio-capillary fibrosis" as due, so far as the *arterioles* are concerned, to changes produced in the perivascular lymph canals by the method of microscopical preparation, viz., treatment of the fresh vessels with glycerine, camphor-water, and acetic acid. By this method the boundary membrane of the lymph space is rendered more distinct, and opacities or striations caused by bendings or duplicatures of this extremely delicate membrane may readily be mistaken for pathological conditions. The indistinct nuclei and cells, which *Gull* and *Sutton* claim to have found in the otherwise homogeneous substance, *Ewald* maintains were merely lymph corpuscles, such as are not infrequently met with in the lymph spaces. The alteration noticed in the *capillaries* by *Gull* and *Sutton*, and which they describe as "thickening by a granular substance," our author regards as devoid of special clinical significance, since the same appearance is observed in connection with a variety of dissimilar affections. His own observations, therefore, afford no support to the hypothesis of a special form of vascular disease—the arterio-capillary fibrosis of *Gull* and *Sutton* which underlies the development of any form of Bright's disease.

On the other hand, he agrees with *Johnson* as to the very general presence of *muscular* hypertrophy of the arterioles when the renal disease is associated with cardiac hypertrophy. In such cases he found that the normal relation of the lumen of the arterioles to the section of their walls (1 : 0.1–0.3) was changed to the proportion 1 : 0.5–1.2, and that this abnormal thickness of the coat of the vessels depended upon enlargement of the muscular fibres, that is, upon a simple muscular hypertrophy without hyperplasia, or new formation of young elements. Except when atheroma, endarteritis, or calcification of the arterioles was present, no change other than that just described was detected.

With respect to the relative frequency of lesions of the vascular apparatus in chronic interstitial and in chronic parenchymatous nephritis, *Ewald* came to the following conclusions. In almost all cases of cirrhosis of the kidney, muscular hypertrophy is present in the heart and vessels. In mixed forms of renal disease where the interstitial process predominates, two-thirds of the cases present both cardiac and arterial hypertrophy, one-third cardiac hypertrophy only; on the other hand, when the parenchymatous changes prevail over the interstitial, all the cases present cardiac hypertrophy without any similar change in the vessels. In pure parenchymatous nephritis no vascular changes were observed, and less than a third of the cases exhibited any cardiac hypertrophy.

Ewald's analysis of his cases shows, moreover, that the cardiac hypertrophy in Bright's disease is not so exclusively confined to the left ventricle as is commonly supposed, as the right ventricle was found hypertrophied in about half the cases. This conclusion agrees with Bright's original observation, as well as with the later opinions of *Traube* and *Galabin.* The hypertrophy of the right ventricle *Ewald* regards as secondary to that of the left, the sequence being here similar to that observed in valvular defects where the compensatory hypertrophy of one ventricle ultimately extends to the other.

As regards the relation, in point of time, between the vascular and the renal changes, *Ewald* entertains no doubt of the precedence of the latter. The arterial lesion, which is essentially distinct from the degenerative changes due to age or other general causes, is frequently met with, as *Dickinson* has shown, after acute nephritis from cold and scarlet fever in childhood and adolescence, therefore at a period of life when degenerative lesions from constitutional causes must necessarily be rare. The order of events, according to *Ewald,* may be traced as follows: Under the influence of the renal disease, blood-changes are developed which increase the resistance in all the capillaries of the body. As a result of this obstruction to the circulation, the general arterial tension is raised, leading to hypertrophy of the heart. The muscular hypertrophy of the arterioles is secondary to that of the heart, and is produced as a direct result of the excessive vascular tension and abnormally forcible action of the heart. That increased arterial pressure is the main factor, however, appears from the absence of vascular hypertrophy in the renal affections complicating heart disease, in atheroma of the large vessels with cardiac hypertrophy, and in the so-called heart defects.

With respect to the *nature* of the vascular lesions in chronic Bright's disease, *Dickinson* (l. c.) takes the middle and probably the safe ground, viz., that the lesion in the arterioles consists partly of muscular hypertrophy and partly of fibrous changes. The latter form of thickening he found in specimens which had been prepared in various ways, showing that the appearances described by *Gull* and *Sutton* were not due to the reagents employed by them. Both forms of arterial change were observed, not only in renal cirrhosis, but also in chronic parenchymatous nephritis, in lardaceous disease and in calculous degeneration of the kidneys. That the changes in question are not due to a general morbid condition, but rather to the renal disease itself, *Dickinson* thinks may be fairly conceded from the fact that the same cardio-vascular lesions are often found in children dying from acute nephritis within four or six weeks after the onset of the disease.

Whatever may be the final outcome of this controversy—whether muscular hypertrophy or fibroid thickening, or a combination of both processes be ultimately established as the essential vascular lesion in chronic Bright's disease—probably the most fruitful result of the prolonged argument will be found in the more general recognition of the wide-spread constitutional character of the pathological changes which have been grouped together under the general term "renal cirrhosis." One of the chief merits of the theory presented by *Gull* and *Sutton* is that it enforces this fact, and directs attention to the *systemic* changes which, from a diagnostic point of view, are frequently more important than the purely renal symptoms. In a series of recent papers, *Mahomed* (l. c.)

forcibly presents this view, and shows that in the increased arterial pressure of chronic Bright's disease we possess the most certain means of detecting the earlier stages of the affection.

> "It has long been a well-known fact," he says, "that high pressure exists in the systemic circulation both in acute and chronic Bright's disease, but it has been very generally believed that this high pressure is produced by the impeded circulation of poisoned blood, and that this poisoned condition of the blood is due to the imperfect elimination of the excrementitious material by the kidney. Thus the sequence is supposed to be: first, diseased kidney; second, retained effete material in the blood; third, impeded circulation; fourth, the cardio-vascular changes characteristic of Bright's disease. This view makes the kidney changes primary, the cardio-vascular, secondary. I have tried, however, to prove that this sequence of events should be reversed; namely, that a poisoned condition of the blood is the primary condition, that this produces an impeded circulation through the capillaries, and subsequently the cardio-vascular changes, while the bad blood produces a congestion of the excretory organs, that is of the skin, mucous membranes, and kidneys, but especially of the latter, and that it depends upon the nature and intensity of the blood poison whether this congestion is acute or chronic.
>
> "The arguments upon which this hypothesis has been based are as follows: first, that high arterial pressure is found to exist before any sign of failure of the kidneys to perform their work occurs. Second, that certain poisons are known to produce kidney disease, and that these poisons produce high pressure in the arteries, while no symptoms of kidney failure are discoverable; on the other hand, the kidneys are often found to be excreting rather more than their usual amount. Third, the condition of high pressure is found to occur in some young people, in all respects perfectly healthy, but liable to a certain class of petty ailments; such patients very often have a family history of gout or Bright's disease, and if they live long enough will almost inevitably develop it themselves. Fourth, far from the kidney disease being the primary condition, I find that patients with primary kidney disease, such as is seen in surgical kidneys or scrofulous kidneys, even of the most advanced nature, do *not* have high pressure in their arteries, while patients with acute Bright's disease, if the poison be acute and temporary, may lose all signs of high arterial pressure during their recovery, even at a time when their kidneys are manifestly crippled, the urine being albuminous, and the solids deficient in amount."

Mahomed shows, moreover, by the evidence of the pathological records of Guy's Hospital during 1877, 1876, and the first half of 1875, "that by far the larger proportion of deaths from chronic Bright's disease occur from failure, not of the kidneys, but of other organs; not only do such cases die with other symptoms than those of kidney disease, but the real cause of death is overlooked." A large proportion die from their cardio-vascular changes.

Analysis showing the clinical aspects of 100 *cases of granular kidneys.*

I. Cases presenting the symptoms of diseases other than Bright's disease. *Red granular kidneys found without epithelial excess:*

Symptoms produced by cerebral hemorrhage,		15
those of heart disease with valvular murmurs in,		17
Of these no valvular disease existed in,	13	
Rheumatic valvular disease occurred in,	4	

those of lung disease,		18
Of these severe bronchitis and emphysema in,	11	
Pleurisy and pneumonia in,	7	
those of sundry other medical diseases,		11
" " surgical "		13
		74
II. Cases presenting some of the ordinary symptoms of Bright's disease. *Mixed granular kidney found with epithelial excess.*		
Symptoms of Bright's disease occurring before death, chiefly acute,		26
		100

"Of these 100 cases we see that 17 died with all the ordinary symptoms of heart disease, and 15 died of apoplexy, making 32 deaths directly due to cardio-vascular changes. Besides these, in the 13 who died from surgical diseases, a good number died more or less directly from their failing hearts, degenerate vessels, and ill-nourished frames. There remain 11 who died from other medical diseases, and 18 who died from lung disease, making up the 74 deaths from failure of other organs than the kidneys. In a very large proportion of these 74 cases albuminuria was absent; when present it appeared to be due to venous congestion secondary to lung or heart disease; renal dropsy was always absent. Thus, only 26 out of the 100 met their deaths from failure of the kidneys, and all those who presented the clinical signs of kidney disease exhibited after death acute or epithelial changes in these organs; and I maintain that it is only such changes that can produce the ordinary symptoms of kidney disease, namely, albuminuria and dropsy. But Bright's disease includes more than this. It appears to me that chronic Bright's disease is caused by a condition of the blood or tissues which may be either hereditary or acquired; it may be regarded as a diathesis the existence of which can be recognized by the condition of high pressure occurring in the systemic circulation, produced by the increased resistance to the circulation of the poisoned blood in the tissues. It produces extensive anatomical changes throughout the body, but especially in the cardio-vascular system, the kidneys, mucous membranes of the lungs, and gastro-intestinal canal, and in the skin. The morbid changes are not necessarily present in any *one* of these tracts, except, perhaps, the cardio-vascular; though usually visible in all it may fall with unusual severity upon either. It reveals itself during life by functional disturbance of one or all of these organs, but the disorder of any one may afford the chief clinical characteristics of the disease, and lead to an incomplete diagnosis in which its true nature is overlooked."

After this general definition of his views upon the essential nature of chronic Bright's disease, *Mahomed* proceeds to describe the signs by which the existence of high arterial pressure can be recognized. The most certain indications are, of course, afforded by the appearances of the sphygmographic pulse-tracing. Increased arterial pressure may be inferred if any part of the tracing rises above a line drawn from the apex of the upstroke to the bottom of the notch preceding the dicrotic wave; or, if the period occupied by the portion of the tracing which corresponds to the systole, viz., from the commencement of the upstroke to the bottom of the dicrotic notch, as measured on the base line, be much more than two-fifths of the whole tracing. The height of the dicrotic notch is also a good gauge of pressure; the higher it is from the base line the higher is the pressure, the nearer it approaches the line the lower is the pressure.

On palpation, the qualities which distinguish the pulse of high pressure are abnormal *length*, *persistence*, and *hardness*, but these characteristics may not all be present at the same time. The quality of *length* is due to the slow, labored contraction of the ventricle in its efforts to overcome the increased arterial resistance, while the *persistence* is explained by the permanent over-distention of the arteries, preventing their normal diastolic collapse. Such arteries often appear to be thickened, but the deceptive sensation communicated to the finger disappears when the vessel is examined after complete closure by compression above the point of observation. Persistence is, however, not invariably a sign of high pressure, for it is often met with in relaxed arteries through which a large but easily and rapidly flowing blood-stream is coursing. The latter condition may be distinguished by the shortness of the pulse and the absence of the heaving character of the high pressure pulse. *Hardness* or *incompressibility* is less constant as a symptom of high pressure, and is directly connected with the more powerful contraction of the heart. It may be absent, however, in vessels which are actually over-distended, but present a small, weak, easily compressible pulse in consequence of failure of the heart. The undue length and latent pushing character of such a pulse can generally be recognized by firm compression of the vessel for some little time.

In doubtful cases, further evidence may be obtained from an examination of the heart. Apart from the evidence of enlargement, other conditions are often sufficiently characteristic, namely, accentuation of the second sound in the *aortic* and not in the pulmonary area, and a reduplication of the first sound, though the latter is less constant; indeed, the first sound may be absent or nearly so if the nutrition of the heart is bad, though high pressure exists to an excessive degree.

The existence of an *habitual* increase of arterial pressure *Mahomed* regards as a matter of the highest importance, and as indicating a certain constitution or diathesis, with certain well-defined tendencies to disease, especially to chronic changes in the kidney.

> "People who are subjects of this diathesis frequently belong to gouty families, or have themselves suffered from symptoms of that disease; in others it may be acquired, and frequently results from lead- or alcohol-poisoning, or takes its rise in pregnancy or scarlatina. In these cases of acquired poison the disease commences frequently in an acute form. In yet other cases there is no distinct poison to be traced; it would rather appear to result from forms of indigestion and malassimilation. . . . The symptom of high pressure occurs very early in life; I cannot say how early. . . . Let it be clearly understood, the existence of this abnormally high pressure does not necessarily mean disease, but only a tendency to disease. It is a *functional* condition, not necessarily a permanent one, though it is generally more or less so in these individuals. These persons appear to pass on through life pretty much as others do, and generally do not suffer from their high pressure except in their petty ailments upon which it imprints itself; these mostly belong to one type, and are generally very greatly relieved by a purge or

a little dieting. In other words, their arterial pressure rises at these periods and calls for treatment. After these little attacks, their pressure often falls, and remains low for a time; gradually, however, it again commences to rise, attains too great a height, and they have another break-down. These breakdowns may be of more or less severity and frequency, according as the diathesis is more or less strongly marked; perhaps they consist of only a little "out-of-sortishness," sometimes severe headaches, often hemicranial, menorrhagias in females, epistaxis in males, temporary albuminurias or hæmatinurias, palpitations, breathlessness, sleeplessness, or the reverse, loss of memory, various neurosal or mental disorders, severe dyspepsias, constipation, or some such troubles; if more severe they may take the form of an attack of gout, or acute Bright's disease, or of bronchitis. But the attack passes off and things remain much as before. As age advances, the enemy gains accessions of strength; perhaps the mode of life assists him, good living and alcoholic beverages make secure his position, or head work, mental anxiety, hurried meals, constant excitement, inappropriate or badly cooked food, or any other of the common but undesirable circumstances of every-day life, tend to intensify the existing condition, or, if not previously present, perhaps to produce it. Now, under this greatly increased arterial pressure, hearts begin to hypertrophy and arteries to thicken; what had previously been a functional condition tends to become more and more of the nature of an organic one. Breakdowns are now, more dangerous, they happen much as before, but more serious ones begin to appear. The individual has now passed forty, perhaps fifty years of age, his lungs begin to degenerate and become emphysematous, he has a cough in the winter time, and gradually drops into a condition of chronic bronchitis, his right heart dilates, and his condition becomes more or less mixed in the aspect it presents to us, but by his pulse you will know him. Or again his symptoms take another line: his heart fails him, it can no longer perform the high-pressure work demanded of it, it therefore fails and dilates; the individual falls into a bad way; a mitral murmur appears; his pulse becomes weak and irregular, though still *persistent*, and so he will remain until he dies, or is relieved by a timely reduction of pressure, which allows his heart to recover, and sets him on his legs again. These cases are generally regarded as ordinary cases of mitral (or sometimes aortic) disease, but no valvular disease is usually present.

"In another case, the heart may not dilate severely; its hypertrophy, with some amount of dilatation, causes more or less trouble; perhaps he comes under observation for some functional disorder caused by it—one of those exacerbations previously noticed; a little albumen may now be found in the urine, the hypertrophied heart and thickened vessels may be recognized, perhaps some hemorrhages seen in his retina, and he is immediately claimed as a case of chronic Bright's disease. The kidney may have a catarrh and the albumen increase in quantity in the urine, and some dropsy may appear. In other cases, the whole stress of the disease seems to fall on the kidney, and it presents the aspect of acute Bright's disease.

"Yet another class of individuals fail through the arteries. . . . Atheroma is their great enemy; it may attack their aorta or large vessels so badly that they get aneurism, and fall victims to this disease. More commonly it causes general aortitis deformans, and creeping from the vessel on to the valves, incapacitates them. The case then appears to be one of aortic regurgitation, and is usually regarded as such. The pulse will usually show the more skilfully hid enemy, whom it is necessary to attack if the patient is to be relieved; it is a pulse of high pressure and is constantly full, although an aortic regurgitant murmur exists, which usually produces an empty or collapsing pulse. On the other hand, perhaps the aorta will more or less escape, while the smaller vessels, especially those of the brain, are the main object of attack. Here we shall have a few warnings; headache, vertigo, epistaxis, a passing paralysis, a more severe apoplectic seizure, and then the final blow. Take the warning which the pulse offers, reduce the arterial pressure, and the patient's life may be prolonged. Or the attack may be more insidious and more difficult to guard against; the atheromatous vessels may become plugged, or by their rigidity may seriously impede blood-supply, and softening of the brain may result. Amidst the general diffusion of atheroma, the coronary arteries may suffer severely; then the stress falls doubly on the badly nourished and overtaxed heart. Attacks of angina warn us of the impending danger, which it is difficult indeed to combat, though temporary relief may be obtained. At last on one occasion the arterial spasm or increased resistance is worse than usual, and the overtaxed heart dies paralyzed by the distention which its degenerate muscle fails to overcome." In still another group of cases, the kidneys fail, and the fatal result is ushered in with symptoms of uræmic poisoning.

It is unnecessary for us to dwell here upon the importance of *Mahomed's* views. If there be a *Bright's diathesis*, as he maintains, the only *pathognomonic* symptom of which is an *habitual* increase in the arterial pressure, and if the presence of such vascular disturbance enables us to predict the development of chronic Bright's disease long before any renal symptoms manifest themselves, the fact cannot be too frequently or too strongly insisted upon. As *Mahomed* points out, there is really nothing new in the supposition that an habitually increased arterial pressure existing in an apparently healthy person, without other symptoms of disease, is really a symptom which either indicates the presence or forewarns us of the approach of chronic Bright's disease. The same opinion has been expressed by *Burdon Sanderson, Handfield Jones, Saundby, Bartels,* and other writers; still no one has elaborated the subject so fully as he, or has so forcibly presented its claims to the consideration of the profession. It is quite possible that he may have exaggerated the rôle which conditions of blood-pressure play in this pathogenesis of Bright's disease; if so, the error is, at all events, in the right direction, and future criticism, even if it modify his theory as to the nexus of events, will at least award him the credit of having presented, more forcibly than any other writer, the great diagnostic value of increased arterial tension as an early symptom in renal cirrhosis.

In a recent article, entitled "Researches on the State of the ganglionic Centres in Bright's Disease" (l. c.), Drs. *Da Costa* and *Longstreth* present the results of an extended investigation of this subject. Whether the lesions they have discovered in the renal plexus prove to be merely a part of the general degenerative changes in Bright's disease, or are finally demonstrated to have an etiological bearing upon the renal process, as they claim, their contribution will, in either case, be welcomed as one of great value to the study of this complex disease. While an examination of the nerve-trunks supplying the kidney gave no satisfactory results, the nerve-centres, that is, the ganglia of the renal plexus, which preside over the innervation of the kidneys, presented changes which appeared to bear a direct relation to the degree of alteration in the kidneys themselves. These changes were most noticeable in the type of disease called interstitial nephritis, or contracted kidney. Incipient changes were also observed in acute and chronic parenchymatous forms of the disease, but how far these varieties are dependent upon ganglionic changes must be decided by future research. In advanced interstitial disease of the kidney, well marked ganglionic lesions were found: connective tissue proliferation, both in the capsule and in the interior of the nervous mass, fatty degeneration and atrophy in the ganglion-cells, and muscular hypertrophy and increase of connective tissue in the arteries, with collapse and closure of their lumina. The interpretation of these ganglionic lesions is difficult. Are they the consequence, concomitant, or the cause of the renal lesions? That they are not the consequence our authors think is proved by the fact that the alterations are discernible in the ganglia before any tissues except the kidneys are markedly affected; but whether these changes are concomitant or cause is not so readily decided. Our authors incline to the opinion, however, that the ganglionic changes are primary; that is, that the unknown specific cause of Bright's disease, whatever it may be, acts first upon the ganglionic centres in the renal plexus, and that through these are secondarily developed the renal changes by means of faulty innervation of the kidneys.

AMYLOID DEGENERATION.

BIBLIOGRAPHY.

LECORCHÉ, M.: Traité des maladies des reins, etc. Paris, 1875. Dégénerescence amyloïde, p. 669.—FÜRBRINGER, P.: Zur Diagnose der amyloiden Entartung der Nieren. Virchow's Arch., 1877, LXXI., 400.—DICKINSON, W. H.: On lardaceous Disease in Reference to its Anatomical and Pathological Relations. Discussion at London Pathological Soc. Med. Times and Gaz., 1879, I., pp. 306, 408, 435, 465.—LITTEN, M.: Berlin med. Wchschr., June 3d and 10th, 1878.—Charité-Ann., Berlin, 1879, IV., 170.—BUDD, GEO., JR.: Amyloid Degeneration. Lancet, 1880, I., pp. 322, 487.

The well-known theory of Dr. *Dickinson*, in regard to the nature of the amyloid substance, viz., that lardacein consists of fibrin which has been deprived of the alkali associated with it in the natural state, has never been accepted as a wholly satisfactory explanation. That the hypothesis rests upon very defective evidence has been recently shown by Dr. *Geo. Budd, Jr.* (l. c.) In the first place, we have no conclusive proof that lardacein is a *nitrogenous* body, as the analyses of *Kuehne* and others seem to have demonstrated. Lardacein is exceedingly intractable to digestive processes, and is therefore very difficult to separate from the normal nitrogenous tissues with which it is associated. This difficulty is increased by the fact that lardacein is deposited, not only on the interior, but also on the exterior of the cell-walls of the organ affected, so that the mass, when subjected to destructive analysis, will yield nitrogen derived from the cell-remains incarcerated therein. In the absence of any means by which the cell-remains can be separated completely, the inference that lardacein is a nitrogenous body must be regarded as unwarranted. Furthermore, *Dickinson* rests his argument that lardacein is *dealkilized* fibrin (acid-albumen) mainly upon the fact that the brown coloration produced by iodine vanishes on the addition of an alkali, and reappears on treatment with an acid. These changes are, however, susceptible of another explanation, viz., that the addition of a free alkali produces a colorless iodide, which is again decomposed with evolution of free iodine on subsequent addition of an acid. In proof of this explanation several experiments are cited, of which we have space only for the four that are most conclusive. 1. The blue color of a solution of iodide of starch disappeared on addition of a few drops of liquor potassæ, and was restored by addition of an acid. 2. A thin microscopical section from a waxy spleen was steeped in a solution of potash for four hours, and then immersed in pure water for a few seconds to remove the free alkali. On treatment with iodine, it at once exhibited the usual mahogany-red reaction. Free alkali, therefore (caustic alkalies which dissolve and decompose lardacein being, of course, excepted), has no action on lardacein which interferes with the iodine reaction. 3. "The same section, which had been already stained with iodine, was treated with a few drops of the alkaline solution, in which it had floated for such a lengthened period; the coloration vanished. Its disappearance, then, is due to action of the alkali upon the iodine." 4. A quantity of lardacein was dissolved in a strong solution of ammonia, and the resulting liquid subjected to evaporation at a low heat. A portion of the scum, consisting of precipitated lardacein, which formed after twenty-four hours, exhibited the usual reaction with iodine, disproving *Dickinson's* theory of acidity, since the reaction appeared even after prolonged treatment with ammonia.

Albuminuria is regarded by all writers as one of the most constant symptoms of amyloid degeneration of the kidneys, but how far the absence of this symptom can be regarded as contra-indicating the existence of this lesion seems to be a matter of dispute. *Lecorché*, for example (l. c.), maintains that albuminuria does not occur in pure amyloid degeneration, but is noticed only when this lesion is complicated by parenchymatous or interstitial nephritis, and is therefore due to the latter processes. He gives no reason for this opinion, which is certainly opposed to the view commonly held, viz., that amyloid degeneration increases the permeability of the affected vessels. That the absence of albuminuria in lardaceous disease of kidneys, even for a considerable period, may occasionally depend upon another cause, is shown by some cases reported by *Litten* (l. c.). In these cases, albumen was absent from the urine for weeks before death, although marked amyloid changes were found at the autopsies. Injection of the kidneys showed that the affected glomeruli were impervious to the passage of the fluid, while the injection freely entered the glomeruli which were healthy or merely exhibited incipient amyloid changes. If the escape of albumen in albuminuria takes place from the Malpighian tuft, as some recent observations appear to show, the above observations may explain the more or less prolonged absence of albuminuria in isolated cases.

TREATMENT OF BRIGHT'S DISEASE.

BIBLIOGRAPHY.

FUCHSINE IN ALBUMINURIA. FELTZ: Gaz. hebdom. de méd., No. 25, 1876.—FELTZ et RITTER: Rév. méd. de l'Est, 1876, VI., 10, 294; 11, 325; 12, 360. Gaz. des hôpitaux, No. 50, 1877. La France méd., No. 42, 1879.—BOUCHUT: Gaz. des hôp., 1877, No. 50; 1878, No. 43; 1879, Nos. 8, 9.—DIVET: Thèse de Paris, 1879: No. 320.—DIEULAFOY: Gaz. hebdom. de méd., 1879, Nos. 30, 32.

BRUNTON, T. L., Arsenic in Albuminuria. The Practitioner, 1877, XVIII., 427.—DUJARDIN-BEAUMETZ: Inhalations of Oxygen in Albuminuria. Gaz. hebdom., 1879, No. 3, 41.—SOUTHEY, R.: Drainage Tubes in Anasarca. Lancet, 1877, 1, 649.—WICKERS, H. A.: On the Surgical Treatment of Anasarca. Med. Times and Gaz., 1879, 1, 7.

MORPHIA IN BRIGHT'S DISEASE. LOOMIS, A. L.: Lectures on Diseases of the Respiratory Organs, Heart, and Kidneys. N. Y., 1875, p. 450.—PARK, R.: The Practitioner, 1875, 241.—POTTER: Boston Med. and Surg. Journ., 1875, May 27th.

BROWN, F. J.: Arrest of Convulsions by the Sinistro-lateral Posture. The Practitioner, 1876, 292.—BAHNSON: The same subject. Virginia Med. Monthly, 1877, No. 7.

JABORANDI OR PILOCARPINE IN BRIGHT'S DISEASE. COUTINHO, S.: Note sur un nouveau médicament diaphoretique et sialagogue, le Jaborandi de Brésil. Gaz. hebdom. de méd. et de chirurg, 1874, No. 15, 239.—BALL et HARDY: Gaz. méd. de Paris, 1874, Nov. 21st.—ROBIN, A.: Gaz. hebdom., 1874, No. 20. Journ. de thérap , 1874, 75.—RINGER, S.: The Practitioner, 1874, XIII., 387.—RINGER, S. and GOULD, A. P.: Lancet, 1875, I., 157.—STUMPF, M.: Arch. f. klin. Med., 1875, 255.—RIEGEL: Berlin klin. Wochschr., 1875, Nos. 6, 7, 46, 47.—PURJESZ: Berlin klin. Wochschr., 1875, Nos. 34, 35.—KAHLER und SOYKA: Centralblatt f. d. med. Wiss., 1876, No. 31. Arch. f. experiment. Path. u. Pharm., 1877, VII., 435. Prager med. Wochschr., 1877, 33.—LANGLEY: Journ. Anat. and Physiol., 1876, 187; 1877, 178.—WEBER: Centralbl. f. d. med. Wiss., 1876, No. 44, 769.—SCOTTI: Berlin. klin. Wochschr., 1877, No. 11.—LEYDEN: Idem, July 2d and 9th.—TYSON: Amer. Journ. Med. Sc., 1877, 2, 132.—JACOBI, A.: New York Med. Journ., 1878, I., 177.—DEMME: Centr. Zeitung f. Kinderheilk., 1878, 3.—BŒGEHOLD: Deutsche med. Wochschr., 1878, No. 49; 1879, No. 26.—VON BRUNN: Idem, 1879. IX., 100.—ROKITANSKY: Ber. d. naturw. med. Verein. Innsbruck, 1879, VIII., 122.—BARKER, FORDYCE: Trans. Med. Soc., N. Y., 1879.—HARTZELL, M. B : Med. Record, N. Y., 1879, XV., 358.—PŒHL, A.: Untersuchung der Blätter von Pilocarpus officinalis (Jaborandi) in pharmacognostischer und chemischer Beziehung. St. Petersburgh, 1879; a very complete monograph.

FUCHSINE (hydrochlorate of rosaniline). Some recent observations appear to show that this substance possesses the property of arresting, or at least of diminishing the discharge of albumen in albuminuria. The discovery was made accidentally in connection with an investigation, in France, of the supposed evils arising from adulteration of wine with this coloring matter. In a report on this subject by MM. *Bergeron* and

Clonet it was pointed out that wine thus adulterated was probably harmless, as in one case of cardiac albuminuria the albumen disappeared while the patient was daily using the adulterated wine. This suggestion led to experiments by *Feltz* and others (l. c.), all of whom agree as to the efficacy of the drug in checking or diminishing the excretion of albumen, though occasional failures were noticed. In all the cases evidence of the elimination of fuchsine was found in the rosy-red color imparted to the urine.

Feltz regards it as chiefly indicated in *parenchymatous* nephritis, in which it probably exerts an alterative action upon the epithelium by which it is excreted. He recommends it in doses of fifteen or twenty centigrammes administered once daily for a week, and then discontinued for a few days, as the diuretic action continues for a time after its withdrawal. *Dieulafoy* (l. c.) reviews all the cases previously reported, and gives four new cases of his own. Although he is skeptical as to the value of much of the evidence, he admits that fuchsine has a decided action in checking the excretion of albumen, but doubts whether the thoracic, cephalic or cardiac symptoms are ameliorated thereby. Further investigation is, of course, necessary to determine the actual value of the drug in albuminuria, but even if, as *Dieulafoy* supposes, the action of fuchsine is limited to checking the elimination of albumen, the drug would prove a valuable acquisition in cases where the loss of albumen is so large as to be in itself a source of exhaustion.

ARSENIC IN ALBUMINURIA. Dr. *Brunton* (l. c.) calls attention to the value of this remedy, particularly in forms of albuminuria which depend rather upon a faulty digestion of albuminous matters than upon morbid conditions in the kidneys themselves. In the interesting case which he reports, there had been a history of albuminuria for ten years, without dropsy or other evidence of renal disease. The albuminuria was generally noticed only in the morning after either bodily or mental exertion, or after the ingestion of meats and fats. After mid-day the patient could work, and eat meat or fat, without the appearance of albumen in the urine. The occurrence of albuminuria was generally preceded by symptoms of acid dyspepsia, and the albumen was found to have a high coagulation point (175° to 180° F.). From these facts *Brunton* concluded that the albumen was derived, in part at least, from imperfect products of digestion in the intestines, in the same way as white of egg is excreted in health when a large number of eggs have been eaten. Fowler's solution, given in three minim doses after meals, effected a complete disappearance of the albumen from the urine, and at the same time improved the general nutrition and strength of the patient.

At a recent session of the Société de Thérapeutique of Paris, the administration of oxygen gas in albuminuria was discussed. M. *Dujardin-Beaumetz* reported one case of chronic nephritis in its last stages, in which, after failure of all other measures, inhalation of oxygen produced a disappearance of albumen from the urine for twelve days. Other cases of temporary disappearance of albumen were also reported. M. *Greléthy* observed that at Vichy it had long been the custom to administer oxygen for both diabetes and albuminuria, and that as a rule both were found to be diminished; but the disappearance was not complete, and in all cases was only temporary.

For the relief of general anasarca two new methods of surgical procedure may be mentioned, which appear to be less frequently followed by unfavorable results than the methods usually employed. Dr. *Southey* (l. c.) employs for puncture of the lower limbs a fine trocar and canula, the latter being but little larger than the needle of a hypodermic syringe. The instrument is introduced into the subcutaneous connective tissue of the calf of the leg, and is connected with a rubber tube, which is drawn over the bulbous extremity of the canula, and conducts the serous fluid into a pan beneath the bed. The canula is secured to the limb by a thread and a small piece of adhesive plaster, and is allowed to remain inserted for twenty-four hours or longer. After removal of the canula, the opening closes without ulceration or inflammatory disturbance. In highly dropsical patients several pints may be drained away daily. The advantages of this method are: A single minute puncture for each limb: cleanliness, and the avoidance of maceration and septic processes about the wound; and regulation of the rate of withdrawal of fluid, if necessary, by means of compression of the rubber tube. Still another method is recommended by Dr. *Wickers* (l. c.) as having been used at the Charing-Cross Hospital in nearly fifty cases without ulceration, sloughing, or inflammation. After oiling the legs, and placing a rubber sheet beneath them, twenty or thirty punctures are made with a stout needle or hair-lip pin. Sponges, wrung out of a saturated solution of salicylic acid, are then placed against the punctures, and renewed every two or three hours. Four or five pints may drain away during the first day, and after the drainage has continued for three or four days, the punctures heal kindly. Decomposition of the dropsical fluid is prevented by the salicylic acid, and cutaneous inflammations of a low type are thus entirely prevented.

Until recently, few points in the therapeusis of Bright's disease have been regarded as so definitively settled as the danger of using opiates in uræmia; indeed, the necessity of extreme caution under these circumstances has become almost an axiom in therapeutics. This position has, however, been squarely challenged by Dr. *Loomis*, and as the subject is a very important one, we prefer to state his views in his own words (l. c.):

> "In 1868 I administered my first hypodermic injection of morphine to a patient with acute uræmia. The effects which followed its administration in this case taught me that, in some cases with marked uræmic symptoms, morphine could be administered hypodermically, not only with safety, but with apparent advantage. Since that time I have used hypodermic injections of morphine in the treatment of patients with Bright's disease, especially when the premonitory symptoms of acute uræmia were present, as well as during the active manifestations of uræmic intoxication, and, so far as I am able to judge, its administration has been uniformly followed by good results. In no instance am I aware that I have caused fatal narcotism. From the the histories of quite a large number of puer-

> peral and non-puerperal cases of acute uræmia in which morphine was successfully used, some of which I have reported in the medical journals, I have reached the following conclusions:
>
> "First.—That morphine can be administered hypodermically to some, if not all, patients with acute uræmia without endangering life.
>
> "Second.—That the almost uniform effect of morphine so administered is, first, to arrest muscular spasms by counteracting the effect of the uræmic poison on the nerve-centres; second, to establish profuse diaphoresis; third, to facilitate the action of cathartics and diuretics, especially the diuretic action of digitalis. Thus morphine, administered hypodermically, becomes a powerful eliminating agent. . . . The rules which are to govern its administration are not well defined. My own experience would teach me to give small doses at first, not to exceed ten minims. If convulsions threaten and a small dose does not arrest the muscular spasms, it may be increased to twenty minims, and the hypodermics may be repeated as often as every two hours. It must be given in sufficient quantities to control the convulsions; neither the contraction of the pupils nor the number of respirations is a reliable guide in its administration."

Dr. *Loomis* is not alone in his opinion that the danger of opium in uræmia has been exaggerated, though, so far as we are aware, no one else advocates such heroic doses.* Other writers, as indicated in the bibliography, have obtained equally good results with morphine in acute and chronic uræmia, and we can add our own testimony to the favorable action of morphine in uræmic convulsions and also in uræmic dyspnœa, but we are far from satisfied that such treatment is wholly unattended with risk. Where the source of danger lies we are unable to say. That simple retention of urea in the blood does not necessarily increase the poisonous action of morphia is clear from the evidence already cited, but it is not so certain that there may not be other as yet unknown morbid states of the blood or tissues in Bright's disease which may render the body specially susceptible to this drug. Most of the fatal cases hitherto reported have been cases in which the *uræmic* symptoms were not pronounced at the time the drug was administered, and yet it would be difficult to convince those whose unfortunate experience it has been to encounter instances where profound and even fatal coma has followed even moderate doses of morphine in Bright's disease that the result was not in some way connected with the renal affection. The whole subject needs careful revision; the danger exists beyond question, but what are the indications for guidance can be determined only by future investigation.

Two interesting observations are reported by Dr. *F. J. Brown* (l. c.), showing the effect of the sinistro-lateral posture in arresting convulsions of uræmic or other origin. His attention was first called to this point from his experience of the good effects of this posture in threatened danger from chloroform inhalation, as first pointed out by Mr. *Bader*. In both of the reported cases the convulsions ceased almost immediately after the patient had been turned into this position. Dr. *Bahnson* (l. c.) confirms this observation from his own experience.

---

* If the doses he recommends for the control of uræmic convulsions can always be used with safety (*twenty minims of Magendie's solution every two hours, without reference to the size of the pupils or the frequency of the respirations*) the uræmic state must certainly establish a special tolerance of the drug. Such unguarded advice we cannot but regard as reckless and highly dangerous.

JABORANDI.—This drug, which has long been used as a domestic remedy in Brazil, was first introduced to the general attention of the medical profession in 1874, by Dr. *Coutinho*,* of Pernambuco, who brought specimens of the plant (the leaves and accessories of the Pilocarpus pinnatifolius) to Paris and called attention to its remarkable diaphoretic and sialagogue properties in the *Gazette Hebdomadaire* (l. c.). The extraordinary eliminative action of the drug at once awakened very general interest, and since then a very copious literature has appeared in Europe and this country on the efficacy of jaborandi in various affections, more particularly in the acute and chronic forms of Bright's disease. The subject has been so fully presented in medical journals that only a very brief consideration of it is required here.

Jaborandi may be administered either as an infusion (60 to 90 grains) or in the fluid extract (20 to 60 drops). Its active principle, *pilocarpine* (the *hydrochlorate* or nitrate of pilocarpia), given hypodermically, is to be preferred on account of the smaller dose required, its more rapid action, and the lessened risk of gastric disturbance. The physiological effects may be briefly described as follows: Flushing of the face, sometimes also of the chest; copious perspiration and salivation (five to twenty-seven ounces and upwards); increased lachrymal, nasal, and occasionally bronchial secretions (with cough); sometimes nausea, even severe vomiting and diarrhœa; rarely vesical tenesmus and strangury; dizziness and sense of fulness in the head; at first increased frequency of the pulse and respiration, and heightened temperature, with subsequent lowering of the same; diminished arterial pressure; a sense of warmth immediately after the administration; then coolness, and finally positive chilliness, with, in some instances, symptoms of absolute collapse. Occasionally tension of the accommodative apparatus of the eye is observed, with amblyopic impairment of vision.

The special indications for the use of jaborandi in Bright's disease are naturally connected with its powerful influence upon the secretions of the body. Marked beneficial results have been obtained in the relief of general anasarca, the absorption of pleural, peritoneal, and pericardial effusions, and particularly in uræmia. For the removal of copious effusions, and in urgent cases of uræmic intoxication, large doses may be required (hydrochlorate of pilocarpia one-half to one grain hypodermically), but, as a rule, smaller doses, repeated if necessary, are to be preferred. With large doses the danger of collapse should be borne in mind and guarded against by the administration of stimulants before, or shortly after the dose is given. Even in moderate doses, however, alarming collapse-symptoms are sometimes produced, perhaps from an idiosyncratic susceptibility to the action of jaborandi in certain individuals; indeed, this danger seems to be the chief objection to the drug, and several writers, especially *Fordyce Barker* (l. c.), whose experience with it in puerperal convulsions was far from satisfactory, have expressed serious doubts whether the depressing effect of jaborandi does not more than counterbalance any good results to be obtained by its eliminative action. The mode in which collapse is produced is not very clear. *Leyden* (l. c.) has shown that it probably does not occur from paralysis of the heart. *Langley*, and *Kahler* and *Soyka* have demonstrated lowering of the arterial pressure after the administration of jaborandi, and have therefore regarded the drug as contra-indicated in valvular diseases of the heart and pulmonary emphysema. *Demme*, however, found no ill effects in these affections, and *Jacobi* used it in seven cases of heart-disease without bad symptoms.

---

* Not *Continho*, as some writers have misnamed him.

Jaborandi is unquestionably a valuable addition to our therapeutic resources in Bright's disease. In certainty and rapidity of action it is superior to the wet pack or hot-air bath, but it is occasionally a dangerous remedy, and should therefore be cautiously used.

RHEUMATISM.—ARTHRITIS DEFORMANS.—GOUT.—OSTEOMALACIA. SLIGHT DISORDERS DUE TO CATCHING COLD.—ANÆMIA. ESSENTIAL PERNICIOUS ANÆMIA.—CORPULENCE. SCROFULOSIS.—MALIGNANT LYMPHOMA. IDIOPATHIC ADENITIS.

BY

CHARLES E. HACKLEY, M.D.,

OF NEW YORK,

VISITING PHYSICIAN TO THE NEW YORK HOSPITAL AND TO TRINITY INFIRMARY.

ACUTE ARTICULAR RHEUMATISM, RHEUMARTHRITIS.

BIBLIOGRAPHY.

Senator: 2d edition v. Ziemssen's Encyclopedia, 1879.—E. Besnier: Rheumatism in Dict. encyclopédique par *Dechambre*, IIIe Série, Tome 4e, Paris, 1876.—E. Leludet: Clinique méd. de l'Hôtel Dieu de Rouen, Paris, 1874, page 119. Pathogénie des accidents cérébraux dans le rheumat.—Pye-Smith: Rheumatism and allied diseases, Guy's Hosp. Reports, 1874.—Beneke: Pathogenesis des Gelenkrheumatismus, Berliner klin. Wochenschrift, 1876, No. 12.—F. Trier: Ueber Hirnzufälle beim acuten Gelenkrh. Nach Nordiskt med. Arkiv, ix. H., No. 8, in Deutsche med. Wochensch., 1878, No. 3.

E. Buss: Die antipyret. Wirkung der Salicyl, Centralbl. f. d. med. Wissen., 1875, p. 276, and Archiv f. klin. Med., xv., page 457.—L. Reiss: Ueber die innerliche Anwendung des Salicyl., Berliner klin. Wochensch., 1875, No. 51, and 1876, No. 16.—F. Stricker: Ueber die Resultate der Behandlung der Polyarthritis rheum. mit Salicyl, Berlin. klin. Wochenschr., 1876, No. 6 and No. 8, and Deutsche militärärztl. Zeit., 1877, No. 1.—H. Senator: Ueber die Behandlung der P. rheumat. mit subcutanen Carbolsäureeinspritz., Nachschr. in Betreff der Salicylsäure, Berl. klin. Woch., 1876, No. 6, and Salicin, ein Ersatzmittel für Salicylsäure, Centralbl. f. d. med. Wissensch., 1876, No. 14, and Ueber die therapeut. Wirkung des Salicins, Berl. klin. Wochensch., 1876, March 4th and 11th, April 15th, Oct. 28th. —Schofield: Treatment of acute Rh. by Salicin, Brit. Med. Jour., 1876, June 3d.—Gräffner: Zur Salicyl-Behand. des Gelenkrh., Deutsche Ztsch. f. prakt. med., 1876, No. 23.—Paul: Treatment of Rheumatism by Salicin and Salicylic Acid, Brit. Med. Jour., 1876, No. 25.—Leonhardi-Aster, Jr.: Beiträge zur Wirkung der Salicylpräparate, Deutsche Zeitsch. f. prakt. Med., 1876, No. 23.—G. See: Traitement du rhumatisme, de la goutte, etc., par la salicylate de soude, Bull. de Thérap., 1877, and Paris, 1877.—A. Jacobi: Acute Rheumatism in Infancy and Childhood. New York, 1875.—Brown: Boston Med. and Surg. Jour., 1877.—v. Ibell: Deutsche med. Woch., 1877, No. 40-42.—J. L. Hicks: N. Y. Medical Record, 1878.—Bericht der k. k. Rudolph-Stiftung in Wien, 1877.—de Renzi: Liguria Medica, 1874.—R. Southey: St. Bartholomew Hosp. Reports, vol. xiv.—Heller: Wiener med. Presse, 1875.—Rossbach: Pflüger's Archiv f. gesammte Physiologie, xii.—Russel Reynolds: Brit. Med. Jour., 1869.—Anstie: Practitioner, 1871.—Luton, Bull. de Therapie, 1874.—Copeman: Brit. Med. Jour., 1874.—Badely: Lancet, 1843.—Weissflog: Deutsch. Archiv f. klin. Med., vol. vii.—Da Costa: Amer. Jour. Med. Sciences, 1875.—von Hölder: Berlin. klin. Woch., 1878.—Hüter: Centralblatt f. d. med. Wiss., 1874.—Dieulafoy: Gaz. des Hôp., No. 99.—A. W. Barclay: St. George's Hosp. Rep., vol. viii.—Wm. Squire: Brit. Med. Jour., 1879.—J. Carafy: St. Geo. Hosp. Rep., vol. viii.—Galezowski: Bull. de l'Acad. de Méd., 1877.—W. M. Ord: St. Thomas' Hosp. Reports, vol. viii.

No morbid alteration of the blood, such as is usually termed a diathesis, has been certainly observed in any of the varieties of rheumatism.

In one form alone, polyarthritis, there is probably an actual diathesis, a change in the blood and fluids. Hence the classification of all other forms with this one on the common basis of "rheumatic diathesis" is purely arbitrary.

Even an hereditary predisposition to polyarthritis, which is not yet certainly proved, would give no reason for the title; as it would merely indicate a tendency to polyarthritis, that is, to a single disease among all those classed as "rheumatic."

Therapeutics of the different forms shows their different nature. Salicylic acid is a true specific for rheumarthritis, and furnishes a test as to whether joint affections are of this nature, and not merely related to it by coming under the general head of "rheumatism." Observation has shown that this and some other remedies act specifically only in rheumarthritis, not in the allied forms of gonorrhœal or chronic rheumatism. We may thus determine the nature of a doubtful case by salicylic acid, as we should a doubtful case of malaria by trying quinine, or of syphilis by mercury. Specific remedies do not depend on the names physicians give to diseases or the classes in which they are placed. Diseases which do not belong together are not cured by the same specific, no matter how much they resemble each other, or how well they seem to fit in the same class.

In regard to the ages of patients, *Senator* says most of the cases quoted are from hospitals, and naturally fewer children than adults with rheumatism enter hospital. Among 214 patients treated at the mud-baths of Nauheim there were from

3– 5 years	4	20–25 years	32	40–45 years	11
5–10 "	23	25–30 "	17	45–50 "	5
10–15 "	50	30–35 "	22	50–60 "	2
15–20 "	38	35–40 "	9	62 "	1

Here the percentage of children is probably too high, as children form a large proportion of the patients at Nauheim.

Race seems to have some influence on the susceptibility to rheumatism, in French and English colonies negroes having been found more often attacked than whites (*Boudin*), but more thorough observations are needed to settle this point.

In regard to the different diseases by which acute rheumatism is said to be caused, *Senator* remarks that anæmia is often accompanied by articular pains and even swelling, but that these should not be considered rheumarthritis. Also that a patient with gonorrhœa may have rheumarthritis, but only incidentally. Statements in regard to the inheritance of rheumatism must be carefully considered, as patients are apt to consider all sorts of pains about the joints from which their parents or children suffer, as rheumatic. To mention some of the more recent statistics, in 400 patients *Pye-Smith* found 23 p. c., and *Beneke* in 214 patients 34.6 p. c., in whom members of the same family had suffered from rheumatism.

Not unfrequently (as is claimed especially by French writers, *Bruguières, Villeneuve, Charcot, Verneuil, Potain,* and others) "rheumatism" immediately follows an injury, surgical operation, etc., either in persons who have never previously had rheumatism, or in those who have suffered from previous attacks and where the latent dyscrasia was roused by the injury. Not a few of these cases were probably chronic articular inflammation of simple or deforming nature, where the injury formed a starting point of an insidious inflammation of some neighboring joint, which has been improperly termed rheumatic. In some other cases, probably the mental impression, the fright, or excitement, so common to injuries and operations, may have had some causative influence. In other cases there may have been a genuine rheumatism.

In regard to the dependence of articular inflammation on precedent endocarditis, *Hueter* suggests the possibility of a dilatation of the vessels or openings of the sweat glands, facilitating the absorption of more inflammatory irritants in the tissue and circulation. The circulation in the blood of the irritants received into the body, he thinks, explains the fever, and if we regard the irritants as small granular bodies, their passage into the synovia and retention there, would act on its intima, and result in serous inflammations. This view would place rheumarthritis among the infectious diseases, and if with *Hueter* we consider the small granular irritants as organized bodies (monads, micrococci), this would be one of the zymotic diseases. *Hueter*, of late, also admits that the joint affections are not always the *result* of an endocarditis, but that both may depend on the entrance of micrococci from the blood into the synovia and endocardium. There is nothing to say against the possibility of this, if we regard the injury inducing the joint inflammations as being granular particles, and still more as organized, that is, as micrococci. But as it has not been proved that micrococci are the excitants of the disease in question, there is no necessity for assuming it as true, no matter how well it may agree with the present fashion of ascribing diseases to organized living bodies (micrococci). It is just as proper to assume that the exciting cause is in solution, as that it is corpuscular or granular particles. So to avoid collisions we may say that some cause of injury, whose nature is unknown, is taken into the body, or developed there, which gives rise to inflammation in the joints especially. This, however, is no explanation, it is only a paraphrase of our ignorance of the actual causes of polyarthritis.

We must stop at this point if we do not wish to go beyond actual facts. But it is an unsatisfactory stand-point; and in the hope of future explanations an attempt must be made to find at least a theoretical connection between the causes and the disease, that is, between catching cold and rheumatism.

PATHOLOGY.

Malassez has recently found, by counting them, that the red blood-corpuscles have greatly diminished in numbers. It has not been decided whether the diminution is greater than in other febrile diseases with similar disturbances of nutrition. *Klebs* says there is fatty degeneration of the liver, which is less marked where death has been speedy, more so when death results after the joint affection has receded considerably. He thinks that the absorption of the articular fluids has a deleterious effect similar to that of the absorption of pus.

Drosdoff says the electrocutaneous sensibility to *moist* electrodes is very much diminished over the affected joints. It may even be entirely wanting, and this condition may exist two or three days before pain appears in the joint, and relapses are frequent until normal feeling returns. He also thinks there is less susceptibility to pressure, while the temperature and sense of feeling are increased. *Beetz* also found the electrocutaneous sensibility diminished, while *Abramowski* found it increased to *dry* electrodes.

Severe lesions of the kidneys and urinary passages are exceptional in rheumarthritis. But during the disease, and undoubtedly dependent on it, inflammations of the kidneys (acute parenchymatous nephritis) have been observed by *von Bartels, Dickinson,* and *Hartmann.*

Corne says he has seen nephritis in a later stage of the disease, where it seemed to have a critical significance and to take the place of the disappearing articular inflammation.

Leudet and *Fränzel* have called attention to the fact that *delirium* may be induced by cerebral anæmia, which may depend on simple nutritive disturbances of the cardiac tissue, with or without pericarditis or endocarditis, just as it is by valvular disease (which may induce cerebral anæmia and psychical disturbances without rheumatism).

The theory of *metastasis* of rheumatic affections receives a certain support from the observations that in some cases the swellings of the joints disappear simultaneously with the occurrence of threatening symptoms of collapse, of brain disease, etc. If we do not accept "metastasis" in the old sense of the word, we must regard these coincidences as purely accidental, or explain them otherwise.

The joint affections in rheumatism are always temporary, they come and go in benign as well as in malignant cases. Even in severe cases their disappearance is often the result, not the cause of the malignancy. The heart's action is impaired, the arterial tension diminished, and the inflammatory hyperæmic swelling, the fullness of the tissues, and even exudations may disappear or diminish. Lastly, some of the metastases of former days may have been due to emboli, which may find a fruitful source in the endocarditis which so frequently occurs. For many of the complications of rheumatism, and for their connection with the joint affections, we can find no sufficient explanation.

The joint affection is the essential feature of rheumatism, and where that is not present we cannot diagnose the disease, but may do so from the articular affection without endo- or peri-carditis or other local trouble. Where from catching cold, or other atmospheric influences local diseases occur, we may term them rheumatic (pleuritis rheumatica, etc.), or we may speak of "rheumatic fever," where from similar causes there is fever with very slight organic disease discoverable; but we do not consider it "rheumarthritis" unless one or more joints are inflamed. We have no reliable sign of the identity of this with other rheumatic diseases, that is with other affections due to the same known or unknown causes. Hence till such a sign is found, all other organic affections, no matter how often they occur or how intimately they may appear to be connected, must be regarded as *complications*.

Among the complications endocarditis is more frequent in children, which fact *A. Jacobi* says is due to the greater demands on their cardiac muscles, the circulation being less aided by the action of the voluntary muscles than in adults; and that the *isthmus aortæ*, being smaller than in adults, offers greater resistance to the efforts of the left ventricle.

It is supposed that complications are much less frequent since the use of the salicylates in rheumarthritis. In 38 cases of rheumarthritis *Brown*, of Boston, saw 3 cases of acute cardiac disease under the salicylic acid or salicin, while in the same hospital under alkalies the percentage was 13.07. According to *v. Ibell*, in 75 patients under salicylate of soda, there were 7 of complications, 6 cardiac, 1 pleuritic. Previously in 337 cases there were 59 complications.

Among 1,853 cases in Hôpital St. André, *Gintrac* only saw meningitis twice. Probably some cases of pyæmia with suppurative meningitis have been erroneously considered as rheumarthritis, or an ulcerative endocarditis with joint disease has been acccompanied by suppurative meningitis along with suppurative inflammations of other serous membranes.

The delirium from debility seen after other diseases marked by high fever is rarely seen in rheumatism.

Dr. J. L. Hicks reports a case of acute rheumatism with meningitis. The patient, aged 72 years, had had previous attacks of acute rheumatism, but no evidence of cardiac complication. The range of temperature was not notably high until the last three days of life; beginning at the outset with a daily fluctuation of from 99° to 101°, and gradually rising to 102° in the second week, and reaching 103° for the first time in the sixth, which was not surpassed until eight hours before death, when the temperature reached 104½°. There was no indication of kidney disease. On autopsy: On removing the calvarium, "the superficial vessels were very full, and showed in strong relief against the whitish linear deposits of lymph in which they seemed at places to be imbedded. The layers of the arachnoid were adherent at several places along the line and on both sides of the superior longitudinal sinus, and patches of lymph were scattered at various points over the convexity of the brain surface. Underneath the membranes, and lifting them up from the brain, was a very considerable effusion of fluid and lymph, filling the meshes of the pia mater, and extending between the convolutions. This effusion was most marked upon the summit, though it existed in a less degree at the base, and a very considerable effusion of both fluid and plastic material was found in the fissure of Sylvius. At the base of the brain the vessels seemed less distended with blood than elsewhere. No embolism or plugging of the smaller vessels could be found anywhere by the coarse method of examination pursued. The surface of the brain had a pinkish, cream-colored tint, and seemed rather firmer than usual, and, on section, the brain substance exhibited numerous sharply-defined puncta. Very little fluid was found in the ventricles, but the choroid plexus presented a distended and infiltrated appearance."

A case occurring in the Vienna General Hospital is given as one of rheumatism of the diaphragm (perhaps with doubtful propriety): A muscular butcher, aged 27 years, was seized with violent pains radiating from the pit of the stomach to the back; respiration was quick, short, and superficial, purely thoracic; face congested, but no fever; patient entirely overcome by his suffering; examination of the thoracic organs revealed nothing. A hypodermic injection of morphine relieved the pain at once, and produced a quiet sleep. The following day the patient complained of pain in the right scapular region, but respiration was not interferred with; he was at once relieved by a repetition of the hypodermic, and next day was discharged cured.

Prof. de Renzi records a case of "rheumatic paralysis of the larynx with slight hysteria," cured by faradization of the skin over the larynx with the electric brush.

DIAGNOSIS.

The disease is not apt to be mistaken, unless the joints affected are few and deeply seated; or in small children where the attention is not attracted by the subjective symptoms to the painful parts. Here a diagnosis may be arrived at by excluding other acute diseases, or more especially by the further course, implication of more superficial joints and occurrence of certain complications (as of heart disease).

Gonorrhœal inflammations of joints are characterized by the presence or precedence of gonorrhœa, and generally by limitation to the lower extremity, especially to the knee-joint.

Some articular pains occurring in patients with anæmia, scurvy, purpura, heart disease, etc., are purely neuralgic, without any change or swelling of the joints, which in other cases may be slightly swollen. This swelling may be the more readily mistaken for true rheumatism, as in these affections there is not unfrequently a more or less irregular fever, even without any perceptible local affection, and because the common anæmic murmurs may lead us to believe that there is a heart affection; or an old cardiac trouble may be regarded as a fresh complication of the joint swelling.

It is doubtful if part of these cases should not be counted as rheumatism; but it seems more proper to make a distinction, as they never pass into the more severe forms, do not have the excessive sweating, or the complications of rheumatism. These points, with the history, anæmia, etc., may serve as aids to diagnosis.

RESULTS.

The mortality which, according to the former statistics, was over

3 per cent, since the use of salicylic acid seems less: of 179 patients treated 3 died (*Senator*).

Death is most frequently induced by rapid elevation of temperature, and cerebral disturbance. Topers are especially in danger. Less frequently, while the polyarthritis continues, death results from complications, particularly pericarditis, or endocarditis, or from affections of the respiratory apparatus, or meningitis.

Among rare sequelæ of rheumatism, *Senator* has seen three cases of weakness of the extensors of the thigh remain after inflammations of the knee-joint, perhaps from the inflammation extending to the muscles. These cases were in young men otherwise healthy, and disappeared after a few weeks' use of the induced current. Another case, where there was spasm of the glottis, terminated in the same way. *Jung* describes one case of paralysis of the deltoid as a sequel.

Dr. Reginald Southey gives carefully prepared tables of the relative frequency and mortality of rheumatism at different ages. He considers it desirable to make a distinction between acute continued and acute relapsing forms of rheumatic fever. "Neither the pneumonic or pericardial complications of rheumatism ordinarily elevate the body-heat above 103.5°." "The subjects of the acute continued form are usually well nourished and quickly flushed by their fever; those of the relapsing variety appear out of condition, sallow or anæmic, and although at temperatures above normal, exhibit no cheek flush." The latter furnish most endocarditis; the former most pericarditis and pneumonia.

"A previous attack of rheumatism has very little influence upon succeeding ones."

TREATMENT.

Puerperal patients and convalescents from scarlatina, dysentery, etc., are especially prone to rheumatism, and should be carefully watched till they recover strength.

In regard to the actual treatment of rheumatism, no plan was followed by decided success till in 1876 the preparations of salicin were introduced; and this marks an epoch in the therapeutics. Since then, proofs of its efficacy and of its superiority to all previous plans of treatment have accumulated rapidly, so that now there can be no doubt of its being our most trustworthy remedy in rheumatism.

After *Buss*, in July, 1875, from a few observations, ascribed to salicylic acid a specific influence in rheumatism, and *Riess*, in the following December, asserted that it cut short the disease, *Stricker*, in the beginning of 1876, from 14 observations, claimed an astonishing effect, and an almost absolute certainty of action. Shortly after, *Senator* proposed to substitute salicylate of soda, so as to escape some of the unpleasant effects of the acid.

Shortly after, *salicin* was recommended by *Maclagan*, who had used it since November, 1874, with the idea that rheumatism and malarial fever were allied diseases, and by *Senator*, because salicin is transformed in the body to salicylic acid, and resembles it in reducing temperature.

The numerous reports published since then of the action of these three preparations are almost unanimous in stating that, when given freely enough, they induce a remission in from 24 to 48 hours, or even an entire arrest of the pain, and soon after of the swelling of the joints, as well as of the fever; and that if the medicine be continued in the same or reduced doses, the disease is rapidly cured, and remains so if the remedy be continued for a week or two in smaller doses. The more recent the case when it comes under treatment, the more rapid and perceptible the result; and this is generally more striking in well-marked, feverish, and active cases, with swelling of many joints, than in the insidious cases with less fever, pain, and swelling attacking few joints, but passing from one to another.

Stricker says that in 181 cases, in military practice, he only had 7 failures under the salicylic treatment. This is probably the best result that has been attained, and the cases were treated under the most favorable circumstances: they were all soldiers who came under the care of the physician at the onset of the disease, and they were persons in the prime of life and strength, and under favorable hygienic conditions.

In 51 cases, *G. See* found the fever and pain lessened in 12–18 hours, and convalescence established in 3–4 days. *Brown* found complete removal of pain in 3 days on the average, the shortest time being 12 hours, the longest 15 days.

In 75 cases of *V. Ibell*, the treatment failed 7 times; of the other 68 cases 7 were arrested in 1 day, 42 in 2 days, 12 in 3 days, and 7 in 3 to 8 days. The entire duration of the disease was 8 days in 34 cases, 8 to 16 days in 31 cases.

It has already been stated that, under the salicin treatment, complications are rarer. This is because the disease is cut short before the occurrence of complications, which does not usually take place till the second or third week. If they are already present when the treatment is begun, they are not influenced by it to any great extent, certainly not so much as the joint affections are: at most there is some arrest of the fever.

The action of salicylic acid in rheumarthritis must be regarded as *specific*, wherein it differs essentially from its merely symptomatic action in other diseases. Here it not only relieves a symptom; its action is not limited to a reduction of the elevated temperature, quieting the articular pains, moderating the inflammation, or arresting the perspiration, but it cures the disease as quinine cures malarial intermittent. Its occasional failure does not militate against its specific action, for even quinine is not absolutely infallible. It is not yet settled how numerous are the failures in properly conducted treatment by salicylic acid; experience on this point being still too young to give certain results, and some of the bad results in the early days of this plan of treatment having been due to the fact that the medicine was used in too small doses or in improper form.

Upon what the specific action of salicylic acid depends we do not yet know, nor shall we know till we have discovered the cause and nature of rheumarthritis. Its most prominent peculiarity, which first brought it into use, its antiseptic and antiparasitic action, render it very probable that it destroys certain organized causes of disease (micrococci), but as these have never been discovered in rheumatism, it becomes a question why it does not exercise a similar curative effect in other diseases where an organized excitant, a *contagium vivum*, is much more probable, in the acute infectious diseases, such as scarlatina, measles, small-pox, etc. It would be necessary to accept the further hypothesis that salicylic acid is only active against certain forms of micrococci which are peculiar to rheumarthritis. If we accept the theory of the action of lactic acid, we might try to explain the efficacy of salicylic acid by its power of arresting lactic acid fermentation; but it is hardly necessary to state that the process of formation of lactic acid outside the body cannot be considered identical with that which occurs within, and further that the lactic-acid theory is far from being absolutely proved.

Pure salicylic acid is best given as powder in wafers or capsules, in doses of 0.5 to 1.0 gram every hour or two, so that an adult may take from 6 to 10, or 12 grams daily at the height of the disease, this being followed by copious draughts of water, mucilage, or milk to prevent irritation of the gastric mucous membrane. The pure acid is not so well borne as *salicylate of soda*, which may be given in somewhat larger doses. A solution of 5 to 10 per cent in aromatic water and syrup may be given in tablespoonful doses every hour or two. These large doses are only continued till the severer symptoms, especially the pain, are arrested or alleviated, which is usually in a day or two. Then the doses are reduced, or given at longer intervals, otherwise the patients soon become disgusted with the remedy. The chief annoyances caused by the medicine are gastric disturbances, such as nausea, tension, etc., tinnitus aurium, dizziness, a sort of drunken stupor, or even delirium and great debility, or collapse. *Salicylate of ammonia* has also been recommended, but has no especial advantage.

In some patients, these unpleasant effects of the drug occur early, and are severe, or it may not be borne at all. *Salicin* is especially suited to such cases, as well as for cases where it is to be long continued, after the severity of the attack has been broken by one of the previously mentioned articles. It may, without disadvantage, be given in larger doses, and for a long time without interruption. It is best given as powder in wafers or capsules, in doses of 0.5 to 1.5 every two hours at first, and subsequently less often. It may also be given in pills or solution with acetic acid. To prevent relapses, these salicylic preparations should be continued in reduced doses a week or two after the subsidence of all symptoms.

All former methods of treatment are inferior to this. They are only to be resorted to in the rare cases where the salicylic preparations are not borne; or where, in spite of proper administration, they fail; or where, after the disease has passed its height, certain symptoms arise which do not yield to salicylic acid, and require other symptomatic treatment.

Heller recommends liquor ammoniæ in drop doses in water every hour, claiming that, in mild cases, it gives relief very speedily, and in severe ones in a day or two.

Rossbach's investigations seem to show that the action of colchicum is probably to reduce the sensibility and reflex excitability.

Among other remedies recommended as specifics is quinine, by *Haygarth, Briquet,* and others. But its action is uncertain; it is most efficacious in large doses for high temperatures, but its effect is only temporary. *Russel Reynolds* recommends tincture of muriate of iron, as does *Anstie,* in doses of ♏ xxx. three to six times daily. Cyanide of zinc is recommended by *Lenton* (Bull. de Thérapie, 1874) .15 daily. *Copeman* (Brit. Med. Jour., 1874) revives the praises of tincture of artichokes (Cynara scolymus), which was spoken of by *Badely* in the Lancet, 1843. Benzoate of sodium has sometimes succeeded where salicylate of soda has failed with *Senator;* it is given to the amount of 6 grams daily.

Local applications may usually be limited to enveloping the joint in some light dressing, or putting the limb on a splint in the most comfortable position. If the pressure of the bed-clothes is painful, a cradle made of hoops may be used. Where the vertebral articulations are affected, we are generally obliged to resort to hypodermic injection of morphine, and hasten the subsidence of the inflammation by leeches and mercurial ointment.

The benefit to be derived from the induced current of electricity is still a matter of debate. *Drosdoff* and others claim that it relieves the pain, while *Weissflog* says it increases pain, in the acute stage at least.

The dyspnœa and palpitation that occasionally occur may be treated with mustard-plasters and other derivatives, such as dry cups over the heart, and tincture of valerian 15 to 20 drops every quarter or half hour.

The psychical disturbances, not accompanied by rapid elevation of temperature at the height of the disease, require no special treatment, unless in case of great restlessness and excitement, when we give chloral hydrate and morphine. For "cerebral rheumatism," *Da Costa* (Amer. Jour. Med. Sciences, 1875) recommends ammmonia and stimulants, but we should hesitate about resorting to them if there is any inflammation of the brain, and unless there is threatening collapse.

When inflammation continues about a joint, and the swelling remains after the fever has subsided, we may resort to leeches, mercurial inunctions, blisters, iodine, warm poultices, or wet compresses, to hasten the absorption. In such cases, we may also use iodide of potash, perhaps with the addition of colchicum.

To avoid the development of chronic articular rheumatism, convalescents should avoid taxing the joints that have been inflamed, and protect themselves against exposure to cold and moisture.

Dr. *von Hölder* has experimented with balsamum antarthriticum Indicum. It is used by inunction with benefit in acute and chronic muscular rheumatism, and as an adjunct to salicylic acid in acute articular rheumatism. He is convinced that the inunctions are beneficial, though less strikingly so, in chronic articular rheumatism and arthritis deformans and in the declining stage of gout. Half a teaspoonful of the balsam should be employed at each inunction, which may be repeated two or three times daily.

At Mt. Sinai Hospital, New York, acute articular rheumatism has been treated by packing the patient in blankets wrung out of hot water, and changed as often as their temperature falls. When the rheumatism shows a tendency to recur, the packing is repeated.

Hueter, of Greifswald, published an article, in 1874, in which he advocated the subcutaneous injection of carbolic acid, as a remedy in certain local affections. He uses a solution of 2 parts of carbolic acid in 100 of water. From $\frac{3}{10}$ to $\frac{6}{10}$ of a grain may be injected at once, and repeated every two or three days p. r. n.: but little pain is caused, and no swelling follows. The solution is supposed to be absorbed by the lymphatics. Injections may be made in the immediate vicinity of the joint.

Dr. *Dieulafoy* injects ten drops of cold water in different points around the joint, with the effect of relieving the pains and enabling the patient to move the joint. The same means may be employed in muscular rheumatism and neuralgias.

Dr. *A. W. Barclay* reports some analyses of urine from cases of acute rheumatism treated by alkalies and quinine and by salicylate of ammonia. He thinks the free administration of alkalies neutralizes excess of acid in the system and induces diuresis. "The number of joints affected, and the severity of the inflammation in each, utterly fail to indicate the amount of alkali necessary to render the urine phosphatic."

"When in rheumatic fever the urine is first of all turbid from the deposit of lithates, no phosphates are precipitated, the acid phosphate of lime being a very soluble salt. But as soon as a large quantity of alkali is poured into the system, this acid phosphate is converted into two neutral phosphates, of which the alkaline phosphate remains in solution and the earthy phosphate is precipitated." From the examinations made while the patients were taking alkalies and quinine, Dr. *Barclay* thinks: "1st. That whatever other benefit may be derived from neutralizing acid in acute rheumatism, its free administration gives rise to diuretic action by which the elimination of waste material is considerably promoted, and that to secure this effect it is desirable to push the remedy till copious phosphatic deposit is obtained. 2d. That the effect of quinine is not in any way injurious, and that it has no influence over the urinary secretion, beyond that of occasionally rendering it less alkaline in its reaction, and that the alkali is not neutralized by any excess of either uric or phosphoric acid, but by some other acid."

Dr. *Barclay* gives salicylate of ammonia in preference to other preparations of salicylic acid. He has found no indication by which to regulate the dose, but gives two-gram doses every two hours till the physiological effects are produced. "With reference to the urinary secretion, one cannot fail to observe that the alkaline treatment has a diuretic power which salicine does not at all possess."

Dr. *Wm. Squire* says: "Rest diminishes waste, prevents pain, induces sleep, and so directly tends to lower fever. . . . Whatever lowers the fever and shortens its duration tends to avert heart-disease."

"The general anæsthesia induced by salicylate of soda has much to do with the antipyretic influence it excites in rheumatic fever." "An increase in the elimination of urea noticed in favorable cases is aided by the salicylate of soda; it acts as a sedative without checking any of the secretions." "As salicylic acid is rapidly eliminated by the kidneys, the doses of it or of salicylate of soda must be frequently repeated."

Dr. *John Carafy* says of salicylate of soda: "With regard to dose and mode of administration, I am convinced that the best plan is to thoroughly saturate the patient with the remedy at the commencement, and then to gradually diminish the dose and lengthen the interval after a distinct result has been produced. Relapses are best avoided by continuing the drug in small doses during convalescence in just sufficient quantity to cause the urine to give a violet tint with perchloride of iron." "It is of the greatest importance that the drug should be perfectly pure. Some specimens smell strongly of carbolic acid, from which salicylic acid is made. All such samples, which also yield a smoky, brownish, or greenish solution, should be at once rejected, as dangerous symptoms of carbolic acid poisoning may result from their use."

Galezowski says, in eighteen cases of rheumatic iritis treated by salicylate of soda, he has obtained amendment of all the symptoms in a few days, and immediate arrest of the pain with speedy disappearance of plastic exudations. In two cases of irido-choroiditis the salicylate arrested the inflammation and pain very promptly. The same was true in inflammation of the sclerotic.

Dr. *Wm. M. Ord* remarks on the use of the "graduated bath" in hyperpyrexia: "The term is used to denote a bath in which the temperature of the water at the time of immersion is at some point between 90° and 100° F., and is gradually lowered by from 25° to 30° during an immersion of half an hour's duration."

Attention is called to two cases of acute rheumatism where the bath was used. In one case, eight 1.25 doses of salicylate of soda at four hours' interval had been given, and produced tinnitus, etc., and the temperature was 106.0° when the bath was used, whereupon it fell to 96.5°.

In the second case, seventeen doses (each 2.) of salicylate of soda were given without perceptible effect, and the temperature was 106.4°, when the bath reduced it to 96.6°. The effect in both cases was lasting, neither again rising over 3°. It is not stated where the thermometer was applied in these cases, but probably it was in the rectum, as its evidence if applied in the axilla would be worthless.

The bath is a sedative to the nervous system, and diminishes the local pain and inflammation. Plunging a patient into a bath at 40° or less causes shock, and may prove dangerous: the graduated bath always seemed to calm the patient. (The same bath is recommended for acute bronchitis.)

GONORRHŒAL RHEUMATISM.

BIBLIOGRAPHY.

SENATOR: 2d edition, v. Ziemssen's Encyclopedia.—PYE-SMITH: Guy's Hosp. Reports, 1874.—BUMSTEAD AND TAYLOR on Venereal Diseases, 1879.—M. MOREL: Rév. des Sciences Méd.—MARTY: Archives générales de Médecine, 1876.—DAVIES COLLEY: Obstetrical Journal of Great Britain, 1878.

The joint affection in gonorrhœal rheumatism is distinguished from rheumarthritis by its symptoms and course, by the absence of complications common to the latter, and lastly by the failure of treatment which is successful in rheumarthritis.

Pretty certainly some cases called gonorrhœal rheumatism have been cases of pyæmia. Some few carefully observed instances of effusions into the knee-joint after prolonged catheterization have been reported. But they do not prove that the affection is a reflex one in the ordinary sense of the term, for the recent and painful cases would give rise to reflex troubles, as in them the irritation would be greatest, and the longer an irritation lasts the less reflex excitement it would cause. In opposition to this the joint affection comes on late in gonorrhœa, usually not for several weeks after the commencement of the disease, that is, at a time when the *pain* of the inflammation has subsided. Finally, the occurrence of actual inflammation from purely reflex cause has never been observed. Those vasomotor symptoms which resemble inflammations and can be regarded as actually *reflex* processes, such as certain hyperæmias, perhaps accompanied by serous infiltration of the tissues, are evanescent as reflex processes usually are, while the joint affections under consideration are characterized by their long duration.

It is incorrect to speak of the reflex nature of the joint inflammation, just as it is when we speak of *reflex* paralyses that are due to a *neuritis migrans*. One of the most probable hypotheses in regard to gonorrhœal inflammation of the joints is, that it is due to a similar process. The fact of its only appearing after a certain time and not at the commencement of the urethral disease, best agrees with the idea that the inflammatory irritation spreads gradually from the urethra to the sacral plexus and spinal medulla and here affects vasomotor or trophic nerves; and thus the joint affection comes about in the same way as in some spinal diseases, and as it is thought to occur in some cases of deforming articular rheumatism.

According to *Pye-Smith*, in 29 cases attacked, the disease was located 14 times in the knee-joint, 10 times in the hand, 3 times in the shoulder, and once each in the hip and elbow.

Treatment by the preparations of salicin, so efficacious in rheumarthritis, is useless in gonorrhœal rheumatism, or at most only temporarily relieves pain, as it seems to do in articular pains from any cause; the same is true of trimethylamin.

It is only inflammation of the urethra that is accompanied by gonorrhœal rheumatism. Balanitis, vulvitis, and vaginitis are not accompanied by it. The connection between the disease of the joints and the urethritis is entirely unknown. Referring it to purulent infection, use of copaiba, exposure to cold, metastasis, etc., is pure hypothesis.

It is not a frequent disease; in 1,912 cases of gonorrhœa, Fournier saw rheumatism 31 times. It is much rarer in women than in men.

It most frequently attacks the knee, but may come in any of the joints, and occasionally occurs in the tunics of the eye. When it affects the joints the inflammation is mostly confined to the synovial membrane, or to the bursæ about the joints. It is not apt to change its seat from one joint to another.

It is usually less painful than rheumarthritis, is accompanied by less swelling and redness, the swelling being mostly due to synovitis. Recovery rarely takes place in less than a month, and the disease may last many months or even years. Restoration to health, though delayed, is usually complete, except in scrofulous subjects.

Fuller says: "In true rheumatism the eye seldom suffers; so seldom that I find no record of any affection of that organ in more than 4 out of 379 cases of acute and sub-acute rheumatism admitted into St. George's Hospital during the time I held the office of Medical Registrar. But in rheumatic gout it is not unfrequently implicated. It was inflamed in 11 out of the 130 cases of rheumatic gout admitted during the same period; and it has suffered more or less severely in 5 out of 75 cases which have fallen under my own care at the hospital."

Gonorrhœal rheumatism occurring in the eye most frequently attacks the sclerotic coat originally, but the vascular connections are so intimate that the inflammation extends to other parts. Where the conjunctiva is implicated, there is a discharge which may simulate gonorrhœal conjunctivitis from direct contact with gonorrhœal matter, but the orbital pains show the implication of the sclera and teach us the real nature of the case.

There are cases of arthralgia occurring in syphilitic subjects which are diagnosed from rheumarthritis chiefly from the history and the concomitant symptoms and lesions. Their course is very sub-acute; there is not the same tendency for so many of the joints to be affected as in rheumarthritis and the systemic condition and sweating peculiar to the latter are wanting. The appropriate treatment is by mercury if the affection occurs at an early stage of syphilis, and by the mixed treatment if at a later stage.

M. Morel states that he has collected all the cases hitherto published —13 in number—of heart affection occurring during the course of gonorrhœa. Of the 13 cases, two were examples of pericarditis and 11 of endocarditis. All the valves of the left side of the heart have been found affected; but the aortic most frequently so. The affection generally shows itself during the course of gonorrhœal rheumatism; but in two cases, it is expressly stated, there was no rheumatism. In five cases the first manifestation of joint affection appeared during an attack of gonorrhœa.

Dr. *Marty*, speaking of gonorrhœal endocarditis, thinks gonorrhœa may be complicated with inflammation of all the serous membranes and may act in a direct way on each of them. Rheumatism is by no means a necessary middle term between the specific lesion and the lesion of the serous membrane, although the cases of co-existence of the two complications are most frequent. The cardiac complications are rare. Of the several orifices the aortic is most commonly attacked. Endocarditis appears to have occurred as frequently as pericarditis, if not more so.

Mr. *Davies Colley* narrates six cases of what he considers a definite joint disease characterized by great pain and tenderness, and more especially by redness and œdema of the soft parts in the neighborhood. In the earlier stages it is accompanied by more or less fever. The diseases with which it may be confounded are erysipelas, phlebitis, or acute suppuration of the joint. The inflammation mostly goes away without suppuration, but the joint remains stiff from fibrous adhesions. The chief seat of inflammation is probably in the fibrous capsule of the joint. The disease in these cases was probably due to uterine or vaginal irritation, often occurring during pregnancy. The only cases of this disease that Mr. *Colley* has seen in the male, occurred in the carpal and tarsal joints. The disease appears to resemble gonorrhœal rheumatism, but there was no effusion into the joints.

The writer of this article has seen one case of such an inflammation of the carpal joint of a female with leucorrhœa, and one of the carpus and another of the tarsus in males with gonorrhœa, with the symptoms and termination above described.

M. Peter, Hôpital St. Antoine, Paris, describes an inflammation of the bursæ and tendons near joints, without swelling or redness, with pain on motion and on passing the finger along the course of the tendons. This disease rarely has cardiac complications, and is seldom accompanied by high temperature.

CHRONIC RHEUMATIC INFLAMMATION OF THE JOINTS.

BIBLIOGRAPHY.

Senator: 2d edition v. Ziemssen's Encyclopedia.—**H. Peters**: Berlin. Klin. Wochenschrift, 1879.—**S. W. Mitchell**: The Relations of Pain to Weather.

One of the points wherein this disease differs from rheumarthritis is the frequency with which it attacks the poorer classes. This also to some extent explains its hereditary transmission, as the unfavorable conditions of life often continue from generation to generation.

Occasionally (especially in women) besides the joints affected in the usual manner, others are diseased as in arthritis deformans. The two diseases run into one another and their distinction may not be possible.

The complication of heart disease, so common in acute polyarthritis, does not occur in the chronic form; or at least, when it is met with, it is a coincidence, or has been developed during a former acute polyarthritis, which has left it, and the tendency to the chronic articular inflammation as sequelæ. There may be an endo- or a peri-carditis, and the same injurious influences may cause an exacerbation of the joint and heart disease; but this does not prove the same connection between them as exists in rheumarthritis.

Dr. *H. Peters*, of Saxony, published some observations on the effect of climate on chronic rheumatism of the muscles and joints. Patients were found to grow worse when the mean temperature fell considerably from one day to another, and when the relative humidity and amount of ozone were high and the wind westerly.

In a paper entitled "The Relations of Pain to Weather, etc.," *S. W. Mitchell* gives his observations on this point, but says: "I found that rheumatic people were unsuited to this purpose, because of the fact that the sensitive ones were too constant sufferers." Hence it would seem that he had not found that connection between rheumatic pains and changes of weather, which is so commonly spoken of by the laity.

TREATMENT.

Remedies having a specific action in rheumarthritis, and which were called "anti-rheumatics," especially the preparations of salicin and trimethylamin, may be entirely inefficacious or of only temporary benefit, for the pain has merely the same effect as in other painful diseases, which are not considered rheumatic, as neuralgias, gout, etc., and cannot be relieved by the treatment which is appropriate as a remedy for pain produced by a different cause.

Irritating rubifacients and plasters act favorably, partly because they induce active dilatation of the blood-vessels and hasten the flow of blood in the joint and its vicinity, which increases the resorption. The same end is better attained by *massage*, which has been much resorted to by the laity, but introduced to the profession by *Metzger*, *Esmarch*, *Billroth*, and others. It is a mechanical manipulation, consisting of kneading, rubbing, and slapping the affected part, combined with active and passive movements, by which the products of inflammation are pressed into the lymphatics and carried off, and the blood-vessels are dilated and resorption hastened.

MYALGIA RHEUMATICA.

Many writers, especially among the French, claim that there is a *hereditary* tendency to "muscular rheumatism;" the term is very indefinite, all sorts of pains being classed under this head, so that we should find that some member of any family had suffered from "pains" of some sort.

Daae (quoted in Virchow-Hirsch's Jahresbericht, 1873) describes as "epidemic muscular rheumatism" a feverish affection with general muscular pains, which started from a farm in Norway and thence spread. This was more correctly described by another observer *Homann* (ibid.) as "febricula contagiosa."

It seems as if the term "muscular rheumatism," must in the future be more restricted and finally abolished. Some of the so-called cases of it might be termed "muscular contractions."

ARTHRITIS DEFORMANS.

BIBLIOGRAPHY.

Senator: 2d edit. v. Ziemssen's Encyclopedia.—G. W. Balfour: Edinburgh Med. Journ., 1876.—E. Ziegler: Virchow's Archiv, 1877.—A. Weichselbaum: Sitzungsb. der Wiener Akad., 1877.—Gaskoin: Med. Press and Circular, 1874.—J. Hutchinson: Med. Times and Gazette, 1876.—E. C. Seguin: N. Y. Med. Record, 1877.—Guneau de Mussy: Clinique Méd., Paris, 1874.

Of late, *Charcot, Cornil,* and *Ranvier* more particularly have studied the anatomical changes in this disease; and have shown that, in the various forms, the *local* changes in and about the joints are the same, and vary in grade only according to the duration of the disease.

But there are differences in etiology and clinical course which justify a division into various forms, although this cannot be done exactly, partly because the etiology is still too uncertain, partly because from the very insidious course the affections have not acquired their typical appearance till a late stage.

It has already been stated that the term rheumatic "diathesis" is indefinite, and can only correctly be applied to acute rheumarthritis, if by a diathesis we understand a condition where some foreign and injurious substance is in the blood and fluids of the body. In all or most of the other forms, the supposition of a diathesis is at present unfounded, and still less is there any ground for believing that a diathesis acting as the cause of arthritis deformans is the same as that of rheumarthritis. The only thing common to all these affections is the obscurity of the causes. In this view, the present form might be termed chronic rheumatic inflammation of the joints, as it is chronic with inflammatory processes. But as every group of diseases depending on doubtful etiological divisions should be reduced as far as possible, and as the affections in question, at least when fully developed, present certain typical peculiarities, it is advisable to separate them from chronic articular rheumatism.

It is evident that this line of separation cannot be very exact: simple chronic articular rheumatism may lead to exactly the same deformities of the joints as arthritis deformans, so that, anatomically, they cannot be distinguished. The same changes may also occur after long-continued *traumatic* inflammations. In all these cases, the changes are mostly limited to a single joint, or, at all events, to a very few, so that the disease may be considered local. If several joints are attacked and become deformed in succession, the diagnosis becomes more difficult, or may be impossible, and it remains a matter of choice whether we shall call the case one of chronic rheumatic inflammation of the joints or arthritis deformans.

Malum coxæ senile has been described as a monarticular or partial form of arthritis deformans, but the cause, the predisposition of advanced age, shows the difference from rheumatic affections, and shows that the trouble is not purely local; and recent investigations have proved that other joints, especially in the spine, may be attacked in the same way. We might ascribe this trouble to a diathesis or dyscrasia, especially to the change in the fluids due to age, which so facilitates chalky deposits, but for the fact that it is not proved that these senile changes alone can induce *malum senile* of the hip or spine.

Gaskoin claims that there is a connection with some cutaneous diseases (especially alopecia circumscripta, lichen, and psoriasis), as he has observed the latter in patients with arthritis deformans, or in those whose parents have had it.

English and French writers believe generally in a hereditary predisposition: they apply the term rheumatism or "rheumatic diathesis" very freely. The form beginning in the small joints especially is said to be due to hereditary predisposition, and to originate particularly from gout in the parents or grandparents.

PATHOLOGY.

Ziegler found that before the cartilaginous covering was destroyed, some bone corpuscles in the bone lying nearest the joint increased in size, the cells lost their processes, the basement substance cleared up, and the spots became cartilaginous. The cells thus transformed from bone corpuscles to cartilage cells acquired nuclei and divided up, so that numerous small enchondromata, not larger than cherries, occurred in the bone.

Then retrogressive changes took place in them; by a sort of mucous softening from within, they were transformed to small cysts.

Dr. E. C. Seguin brought before the New York Pathological Society, Nov. 14th, 1877, two children with arthritis deformans. "The family consisted of four children; of these, three had the disease, one boy and two girls. In all the disease developed itself from the age of 2½ to 4 years. In none of them was there any symptom of progressive anchylosis. The disease commenced in the last phalanges, then attacked the second row of joints, and thence advanced to the wrist, elbow, and feet. There was no difference in the three histories and no difference in the appearance of the three children. The parents were free from any disease. There was no assignable cause for the trouble in the children." They were under galvanic treatment, with possibly some slight benefit. The extreme rarity of this condition in infancy constituted the interest in the cases.

According to *Weichselbaum*, the changes are only a higher grade of simple senile changes. There is a difference between the two forms, namely, that starting from the small joints (polyarticular) and that involving a large joint (monarticular, *malum senile*). In the former, the inflammatory proliferating processes of the synovial membrane predominate; the cells of the latter (as chondroclasta) enter the neighboring cartilage, and gradually cause its atrophy. Then the proliferating synovial tissue gradually causes a union of ends of the bone, whose cartilage has been destroyed, and the joint is partly or entirely obliterated, and becomes fixed. At the same time, there may be fibrillation of the cartilage and exposure and hardening of the bone; but this occurs to a less degree than in the other form. In the latter, this proliferation of the synovia is less prominent, while the fibrillation of the cartilage and hyperplasia of the cartilage and bone about the surface of the joint are more prominent, and the cells of the synovia are transformed to cartilage cells, and cause the nodules which appear.

TREATMENT.

When the disease has once begun, we may try to arrest its progress by improving the hygienic conditions, choice of climate, dwelling, sleeping apartments, occupation, etc.

Next to the employment of iodine comes that of arsenic, which is recommended especially by the English (*Fuller*, *Balfour*, and others). Ten to fifteen drops of Fowler's solution may be given thrice daily after eating.

Where the patients are much run down, cod-liver oil may be prescribed; it is said to improve the general condition and arrest the local symptoms, or even to cause their disappearance in cases not far advanced. In suitable cases, especially if the digestion allows, it is well to combine iodine with the cod-liver oil. All other internal remedies recommended, usually those given for chronic articular rheumatism, are of no use.

Of the warm baths *Gueneau de Mussy* praises those containing arsenic. Massage may also be tried with some hope of benefit.

GOUT.

BIBLIOGRAPHY.

Senator: 2d edition of v. Ziemssen's Encyclopedia.—Hutchinson: Medical Times and Gazette, 1878.—G. Owen Rees: Brit. Med. Jour., 1877.—Robert Halma Grand: Gaz. hebdomadaire, 1877.—B. J. Stockvis: Centralblatt f. d. med. Wissensch., 1875.

Hutchinson says the younger children of a family are more often and more severely attacked by gout than the elder ones, because if the parents have gout, it increases in severity as they grow older.

Among 500 gouty patients, *Durand-Fardel* found only 20 women; among 80 others, *Patissier* found only 2 women.

According to *Garrod*, patients who had previously had rheumatism, when attacked by gout, usually suffered in the joints that had been affected by rheumatism.

Hutchinson says gouty people are generally rheumatic, *i. e.*, they inherit the rheumatic tendency. But the reverse does not hold—*i. e.*, rheumatic people are not necessarily gouty: the gout is something superadded to the rheumatic diathesis. It is dyspeptic and it means mal-assimilation; it has to do with stomach, liver, and kidneys. When a gouty subject has an arthritic tendency, his gout will manifest itself in the joints; if otherwise, his joints may escape and he may suffer from dyspepsia, neuralgia, etc. On the other hand, give a rheumatic subject diseased kidneys and he will very likely have gout. We see then that gout is in relation to the kidneys or, more generally, to increase of waste material. It does not necessarily mean diseased kidneys. It may be the result of altered living or of growing old and consequently taking little exercise.

Rheumatism and gout are so closely allied that if the subject of gout has not had rheumatism, probably his relations have. In both there must be a predisposition before some exciting cause induces a manifestation of it. The rheumatic diathesis means a condition of nervous system which is highly susceptible to weather and external influence; it is therefore climatic. Gout, on the other hand, is dietetic. Gout is frequently attended by disease of the vessels, and there is always a liability to their rupture; hence red patches on the conjunctiva from ecchymoses and epistaxis are not uncommon in gout. A certain kind of retinitis called hæmorrhagic, and characterized by numerous effusions all over the retina, each one being flame-shaped, is quite pathognomonic of gout.

Various skin affections (eczema, acne, psoriasis), are sometimes ascribed to a gouty diathesis; but there is no certain way of telling if this is really the cause. The only certain anatomical evidence of gout on autopsy is the change in the joints. In one case *amyloid degeneration* of the spleen and of the contracted kidneys was found by *Litten*. *Garrod* claims to have found oxalic acid in the blood repeatedly.

Dr. *G. Owen Rees* thinks "that the phenomena of the acute gouty paroxysm are best explained on the theory that the disease is essentially a capillary phlebitis, the venous inflammation being caused by the circulation of a blood-poison," . . . "there must be some condition necessary to the production of gout other than the presence of urinary constituents in the blood, while it must still be admitted that such contamination plays a considerable part in the causation of the disease. We cannot doubt that before an attack of gout occurs, the blood always contains an excess of uric acid."

"*Sir James Paget* has shown that the larger venous trunks are especially prone to disease in gouty persons. If so with the larger trunks, why not so with the capillaries?" "The venous capillaries inflame, . . . severe pain is experienced, which is relieved immediately when effusion and swelling occur."

Although the relation of gout and lead-poisoning is recognized, the cases published are few; Dr. *Grand* adds thirteen.

"Saturnine gout is distinct enough from true gout to deserve a special description. One of the greatest predisposing causes to gout is the prolonged influence of lead; and this form of gout is very rapid. It becomes chronic and causes deformities much quicker than common gout; this is probably due to the retention of uric acid in the organism, and its accumulation due to lack of elimination by the kidneys. . . . Prof. *Charcot* thinks that there is increased formation of uric acid on account of hepatic hyperæmia."

According to accurate examinations of the urine by *Stokvis* in the first two days of the attack, the urea decreased more than one-half (from 26.01 to 11.06), then increased beyond the ordinary amount (to 30.26). The phosphoric acid of the urine was diminished the first day only, was increased the following two days, and fell the fourth day, so that on the average it was less than ordinary, amounting to 0.9184 instead of 1.041 grams. The phosphoric acid in combination was proportionately much more diminished. The alkalies of the urine were diminished the first day, the earths during the whole attack.

Stokvis has also made careful examinations of the state of the phosphoric acid in the urine during the intervals between the attacks. In spite of rich nourishment and considerable excretion of urea (40.23 daily), it was diminished considerably (to 0.688 daily). This diminution particularly affected the phosphoric acid combined with alkaline earths, which disappeared entirely on some days. Consequently the proportion of this part of the phosphoric acid to the part combined with alkalies was as 1:5.7 instead of as 1:2.5. Organic acids, especially citric acid, appeared to diminish the amount of phosphoric acid, while in a non-gouty person it increased it. Mineral acids (phosphoric, muriatic), increased the excretion in gouty persons, but more slowly than in other persons, and in the former the increase was in the earthy phosphates, in the latter in the alkaline."

In the first edition of v. Ziemssen's Encyclopedia sarcosin was spoken of as a remedy which would probably be useful in the treatment of gout; but *Senator* now believes that the suggestion was based on false premises.

Salicylic acid is useful here as in other painful affections of the joints, but in gout the digestive organs are apt to be disturbed, and this disturbance may be increased by the remedy; hence salicin is better than its salts in such cases.

Lallemand's gout specific is said to be prepared as follows (New Remedies, see *Med. Record,* 1879).

℞ Ext. colchici acet.,	
Ext. opii aquos	āā 1.
Potassii iodid	16.
Potassæ acetat	8.
Aquæ destil	110.
Vini alb.	122.

M. S. Twenty drops three times daily.

In the *Jour. de Méd. et de Chir.*, a "physician of forty years' standing" says he has for two years avoided attacks of gout, rheumatism, and sciatica (from which he had previously suffered several times a year), by taking one gram of salicylate of soda with each meal, except during the summer, when he reduces the dose to two grams daily.

OSTEOMALACIA.

BIBLIOGRAPHY.

SENATOR: 2d edition, v. Ziemssen.—RAGSKY: Archiv f. physiolog. Heilkund.—V. LANGENDORFF and J. MOMMSEN: Virchow's Archiv, 1877.—MONDAN: Lyon méd., 1876.—F. NOHL: Ueber Osteomalacia, Diss., Berlin, 1876.—UTZ, as reviewed in Virchow-Hirsch's Jahresber., 1874.—RINDFLEISCH: Lehrbuch der patholog. Gewebelehre.—F. SCHMUZIGER: Centralblatt f. d. med. Wiss., 1875.—BENCE-JONES: Ann. de Chimie et Phar., LXVII.—KÜHNE: Verhand. des naturf. Vereins zu Heidelberg, II.—SPÄTH: Reported by London Med. Record, 1878.—STERNSCHUSS: Med. Chirurg. Centralblatt, 1877.

In this disease the specific gravity of bones (normally, after being cleaned, 1.877, according to *Valentin*) has been found reduced to 0.721.

The disappearance of the chalky or mineral portion of the bone seems to proceed from the medullary cavity toward the surface of the bone; the chalky salts being first dissolved out by some acid from the Haversian canals of the trabeculæ of the spongy substance, and then gradually from those of the surface of the bone.

Mommsen found in his case no resemblance in the structure of the diseased bone to that of one deprived of its lime by acid, but it was more like the bony tissue of young infants as described by *Ebner*. For while in normal bone, that has been deprived of its lime, the striæ of the basement substance are quite broad and of varied refractive power, *Mommsen* found the striæ all fine and of the same refractive power. The Haversian canals were considerably dilated, the normal lamellar system nowhere more complete, and the boundary lines of the different lamellæ (*Ebner's* "cement-lines") rather indistinct. At other points the basement substance was simply composed of long filaments and seemed to be continuous with the lamellæ, transverse striæ gradually appearing among the longitudinal. In some parts the bone corpuscles showed the most varied forms and were grouped irregularly, the basement substance appearing homogeneous or somewhat granular when slightly magnified, but when more strongly magnified showing fine filaments felted out in all directions, and at these points there were numerous perforating filaments (*Sharpey's*) which are not found in corresponding healthy bones. In this case the medulla of the bones showed the different stages of change mentioned above. There were cysts of various sizes formed by absorption of bone elements. The larger cysts were enveloped in a tough, slightly vascular membrane, which had no epithelium within, and numerous pigment granules without. The smaller, indistinctly bounded cystic spaces contained fat-globules, blood and pigment granules, and here and there a bony trabecula. Near the cysts, more rarely in the other bony tissue, there were numerous giant cells in the dilated Haversian canals, especially at the margin toward the bone, which showed the corresponding *Howship's* lacunæ. Besides these, there were in the Haversian canals variously formed round and spindle cells.

Any of the bones may be affected by osteomalacia, even the ossicula of the ear are said to have been softened in the celebrated case of *Mrs. Supiot*. The teeth are probably never deprived of their chalky substance or softened, but they usually become carious and are lost.

Certain kinds of soil and deficient nourishment seem to influence the occurrence of the disease, judging from its endemic appearance in certain districts, and still more from the softening of the bones, similar to osteomalacia, which occurs in animals pasturing on land deficient in lime. According to *Utz* this disease occurs in the Black Forest, where the water contains little lime, and is cured by giving salts of lime. Numerous similar experiences of the deficiency of lime in food have been mentioned in the case of rachitis.

However, it has not been proved, and perhaps is not even probable, that the food of osteomalacic persons is particularly deficient in lime, or more so than that of other persons living under similar circumstances; indeed, the adult body requires but little lime. Hence there must pretty certainly be other causes to blame for the deficiency in the bones of earthy and especially of chalky salts. The soil can have only a remote and mediate effect by influencing the quality of the water and food, which may prevent the restoration of lime that has been lost from the economy by abnormally increased excretion or use. Now this increased requirement of lime may be found in pregnancy (in that of the lower animals as well as in the human being, according to *Utz*) which is one of the great predisposing causes of the disease, on account of the accompanying greater absorption of lime, which is necessary for the growth of the fœtus, this not being replaced on account of deficient supply or from defect of digestion. But in osteomalacia we have no evidence of such defects of digestion as we have in rachitis.

Hence another and more popular view seems more probable; viz., that the loss of lime from the bones in osteomalacia of man is not due to defective supply, but to a solution of the salts that have already been deposited, by abnormal action of some acid, probably the lactic which is suspected of causing rachitis, possibly also of other organic acids and carbonic acid.

The points in favor of the hypothesis of an acid action, and particularly of the effect of lactic acid, are, that the contents of osteomalacic bones have been repeatedly found to be of neutral or even acid reaction, and that lactic acid has been found in them as well as in the urine of patients, which, in one case at least, has been observed to disappear from the urine as the patient improved. On the other hand, it must be acknowledged that experiments for inducing osteomalacia by administering lactic acid have failed, and that no source for abnormal development of lactic acid has been proved to exist in osteomalacia.

Examination of the bones teaches that there is a combination of active and passive changes; at first there is an active hyperæmia with new formation of young cells, giving the changes in the bone a resemblance to those due to inflammation. It is possible that under irritation the medulla of the bones, like the tissue of the spleen (which is of allied nature), may produce in excess certain organic acids, of which only the lactic has thus far been discovered. This hypothesis would be favored by the frequency of the disease during pregnancy and its commencement in the pelvic bones, for during pregnancy there is physiologically a congestion of the pelvic organs and imbibition of the pelvic bones with blood, which may readily increase to an abnormal extent.

Rindfleisch was the first to indicate that solution of the salts in the bones, by excessive development of carbonic acid, might induce osteomalacia. This excessive development he refers to passive congestion in the bones, as he can find no cause for active hyperæmia. In the question at present under consideration, this distinction is of no account, for it has been shown that, in active and inflammatory hyperæmia, carbonic acid accumulates in the bones in excess. This theory is supported by the investigations of *Flesch* and *Tillmans*.

So, at present, we may believe that the solution of the bones in osteomalacia, in the human being, does not depend on insufficient supply of lime or excessive supply of acids to the bones; but that the first change is in the bones themselves, probably hyperæmia and inflammation, as a result of which there is an excessive development in them of lactic, carbonic, and probably other organic acids. The disease may be favored by increased consumption and insufficient supply of earthy salts.

Winckel says that patients with osteomalacia have inclination to muscular trembling and spasms, similar to the fibrillar twitching observed in progressive muscular atrophy. This was noticed more especially in puerperal and nursing women.

Patients early become wrinkled and old-looking, especially when their teeth are carious, as so often happens.

Recent observers have found the urine slightly acid. *Schmuziger* twice determined the degree of acidity of the urine passed in periods of twenty-four hours by a woman with osteomalacia, and on successive days found 1.026 and 0.863 of oxalic acid, which is much less than normal. In regard to the phosphates also, recent investigations have shown that in most cases there is no increase, but on the contrary considerable diminution. The proportion of alkaline earths, especially of lime, is not certainly known, as this would require continuous examination of the urine, from the beginning to the end of the disease, which has not yet been done. It is possible that the excretion varies in different stages of the disease. Perhaps in the commencement, while solution of the bone is going on, an abnormal amount of lime and magnesia may be excreted, subsequently less and still later an abnormally small amount.

In a pregnant female, forty-two years old, in five consecutive days, *Schmuziger* found the amount of lime passed in twenty-four hours to be from 0.0624 to 0.08, or, reckoned as phosphate of lime, 0.1151 to 0.1476. On two days, the earthy phosphates amounted to between 0.2948 and 0.3332 gram. *Langendorff* and *Mommsen,* in the urine taken during twenty-four hours from a thirty-three-year-old man, much debilitated, found 0.15 to 0.225 gram of lime. The amount of all the phosphates once amounted to 1.397 gram.

Schmuziger found no lactic acid in his case; *Langendorff* and *Mommsen* found it in their case, but also found it in the urine of a healthy person. The proportion of the other constituents of the urine (urea, uric acid, chlorine) has been but little investigated; in *Schmuziger's* case they seemed little altered.

Albumen has been repeatedly found in the urine, but there may have been catarrh of the bladder or of the vagina in these cases. In one case *Bence Jones* found an albuminoid substance, which, precipitated by alcohol and dried, dissolved in water, and became gelatinous and was precipitated from an alkaline solution by acetic acid. *Kühne* also found this substance, "hemi-albuminose," in one case, and in *Langendorff* and *Mommsen's* case, instead of the ordinary albumen reaction, the urine gave the same reaction as described by *Bence Jones.*

According to *Gusserow,* the milk of osteomalacic women is abnormally rich in chalky salts; this is denied by *Pagenstecher.* Older authors also speak of salt being excreted through the sweat and saliva. *Pagenstecher* thinks this also occurs through the bronchial and intestinal mucous membranes, and there induces catarrh, while the excretion through the urine is lessened.

Dr. *Späth*, at the Imperial Royal Medical Society of Vienna, described a case of osteomalacia in which he had successfully performed Cæsarean section with extirpation of the uterus. The patient was in her tenth pregnancy, the previous labor having been accomplished by perforating the child's head. She had bronchial catarrh and albuminuria which ceased after the operation; while under observation, subsequent to the operation, no advance of the osteomalacia was observed.

Dr. *Sternschuss* reports the case of a patient, thirty-three years old, in her ninth pregnancy, whom he delivered by perforating the child's head, the pelvis having assumed the heart shape common to osteomalacia.

SLIGHT DISEASES DUE TO CATCHING COLD.

BIBLIOGRAPHY.

E. SEITZ:—2d edition, v. Ziemssen's Encyclopedia, 1879.—W. H. J. WOOD, British Medical Journal, 1876.—J. B. BERKART, Medical Press and Circular, 1878.

If catching cold be due to cooling of the skin, the therapeutic application of cold and moisture to the surface of the body may cause it, but it is less apt to follow the cold bath, douche, etc., on account of the short duration of the application. The local application of cold to the head and limbs seems less often to excite colds than its use on parts which are ordinarily less exposed. Daily experience shows that cold applied to hyperæmic or inflamed parts, or during protracted fever, less frequently causes colds than under ordinary circumstances.

At present the popular belief is that fever is caused by the occurrence in the blood of a pyrogenous substance whose nature is unknown. In this case we must infer that in fevers due to catching cold, this act must induce the pyrogenous blood change, which in its turn excites increased irritation and activity of the calorifacient centres of the nervous system.

Senator remarks that in man suppression of cutaneous perspiration, as appears frequently to be done by inunctions, does not cause serious results as it does in animals, perhaps on account of the previous shearing or plucking of the animals having induced irritation of the skin.

In his article in v. Ziemssen's Encyclopedia *Seitz* only speaks of diaphoresis in the treatment of slight affections due to catching cold; but probably most practitioners in this country have noticed the excellent effects from a full dose of quinine or twenty drops of muriated tincture of iron diluted in water and taken at one dose, to be repeated in two hours. Possibly these act as the hot punch or a full supper does; they are all useful only at the *commencement* of the attack.

Acute naso-pharyngeal catarrh is said to be cured as quickly and certainly by total abstinence from liquids for twelve to sixty hours as by any other means.

"As derangements of nutrition are invariably produced by a *high temperature* of only a short duration, it cannot be doubted that the prolonged action of a *lesser* degree of heat must have similar consequences. Indeed it appears that such is the case in those who most emphatically declare their susceptibility to 'catching cold.' One acquainted with their habits of life will readily conceive that they seldom or never give *cold* the least chance of exerting its injurious influence, but that they suffer from the consequences of overheating. Their vessels are dilated and degenerated; serum and even white blood-corpuscles escape on the slightest provocation. A heated room, the irritating gases—the products of respiration and combustion, such as accumulate in the air of a theatre—and the dust which a current of air, *the draught*, raises from the ground; these suffice to produce the supposed effects of 'catching cold.'"

ANÆMIA, OLIGÆMIA.

BIBLIOGRAPHY.

H. Lebert: Archiv. gén. de méd., 1876.—Hayem: Gaz. méd. de Paris, 1876; Archiv. de phys., 1877; Gaz. méd., 1877; Comptes rendus, etc., Geneva, 1878.—J. Cohnheim: Vorlesungen über allg. Pathol., 1877.—Samuel: Ueber die Entstehung der Eigenwärme und des Fiebers, Leipzig, 1876, and Handbuch d. allgemeinen Pathologie, Stuttgart, 1879.—Wiskemann: Spectralanalytische Bestimmungen des Hæmoglobingehaltes des menschlichen Blutes, Freiburg, 1875.—Buss: Zur antipyret. Bedeutung d. Salicylsäure und des neutralen salicyls. Natron, Stuttgart, 1876; Ueber die Entstehung der Eigenwärme u. des Fiebers, Leipzig, 1876.—Vierordt: Grundriss der Physiologie des Menschen, 1860; Physiologie des Kindesalters, Tübingen, 1877.—Eisenlohr: Deutsches Archiv f. klin. Med., Bd. XX.—Litten: Berl. klin. Wochensch., 1877.—Lépine and Garmont: Gaz. médicale, 1877.—Leichtenstern: Ueber den Hæmoglobingehalt des Blutes in Krankheiten, Vortrag, Tübingen, 1877.—Malassez: Archives de Physiologie, 1877.—A. Schmidt: Die Lehre von den fermentativen Gerinnungserscheinungen u. s. w. Dorpat, 1877.—Immermann: Deutsches Archiv f. klin. Med., Bd. XIII.—Liebermeister: Do., Bd. VIII.—Quincke: Ueber Siderosis, Festschrift, Bern, 1877.—Eichhorst: Virchow's Archiv, Bd. LXX., LXXIV.—Uffelmann: Deutsches Archiv f. klin. Med., Bd. XIV., Bd. XX.; Die Diät in den acut-fieberhaften Krankheiten, Leipzig, 1877.—G. W. Balfour: On the Position and Mechanism of the hæmic Murmur, Lancet, 1877.—Forster: Zeitschrift f. Biologie, Bd. XI.—Immermann: v. Ziemssen's Encyclopedia, 2d edition, 1879.—Panum: Virchow's Archiv, Bd. XXIX.—Paquelin: Comptes rendus du Congrès périodique international de Genève, 1878.—Niemeyer: Text-book of Practical Medicine. Revised Translation, 1880.

The physiological function of the blood is to supply the tissues, from without, with oxygen, water, albumen, fat, carbo-hydrogens, and salts; also to remove the results of regressive metamorphosis by way of the lungs, kidneys, and skin. Two factors render this possible. 1. The red blood-corpuscles, whose hæmoglobin has the special power of carrying the oxygen taken in during respiration. 2. The peculiar physical properties of the plasma, which render the blood capable of circulating and of acting as vehicle for the red corpuscles, as well as of the nutritive and excrementitious substances to and from the other tissues.

The organism is only in perfect health when the blood contains sufficient well-formed and functionally active red blood-corpuscles, and enough plasma of good circulating quality. If these conditions be not fulfilled we have *anæmia* with its symptoms of deficient energy and impaired blood function. It seems due to lack of both of the above constituents; but they probably are not both equally implicated in all cases of anæmia.

Leichtenstern, investigating the amount of hæmoglobin by *Vierordt's* method, found the blood of healthy new-born children to contain the largest proportion. In ten or twelve weeks it sinks to the average of adult life; then it falls and remains low from the sixth month to the fifth year; and the second highest point is from the twenty-first to the forty-fifth year.

Protracted fevers, chlorosis, leucocythemia, and more especially essential anæmia, are accompanied by reduction of hæmoglobin.

Hayem, in oviparous vertebrates, found cells without color, but different from white corpuscles, which he thought developed into red corpuscles, and called them "hæmatoblasts." It is not yet certain whether they are a form of the leucocytes.

The effect of the red corpuscles seems to depend on their number, and a pathological influence may be expected when their number is absolutely too small for the size of the body and the physiological requirements. This condition is called *oligocythæmia* (ὀλίγος few, κύτος cell, αἷμα blood) and is present in most cases of anæmia; examination of the blood shows that the red corpuscles have disappeared from the blood of anæmic patients in greater percentage than the other constituents (leucocytes and plasma). But as the power of the red corpuscles depends on the hæmoglobin, deficiency of the proportion of the latter in the cells would also cause an anæmia, which may be distinguised from the former as an *oligochromæmia* (ὀλίγος little or few, χρῶμα color, αἷμα blood). Both forms would cause symptoms due to deficient supply of oxygen to the tissues.

Besides these two, there are other anomalies of the red corpuscles, such as abnormal smallness of many of the cells (*microcythæmia*) or malformations of them (*poikilocythæmia*).

But anæmia may also depend on changes in the blood-plasma, its conductive power being perfect only when it is present in full amount. Although the amount of plasma depends on that of its chief constituent—water—when its quantity is too small, there is usually a deficiency of all the constituents of the plasma. Cases do occur where diminution of the plasma is chiefly or entirely due to loss of the water, while the other constituents are not affected; this would cause abnormal concentration of the plasma and reduction of the volume of blood (inspissatio sanguinis, anhydræmia, anæmia sicca), such as occurs in cholera and probably in heat-fever or insolation.

More frequently the plasma is diminished, but the concentration is normal, or lessened as compared with certain other constituents of the blood. In all these cases, which are classed as anæmic, there must be a complicated disturbance, all the other constituents being diminished proportionately to the plasma water, or some of them in greater proportion. To understand this modality we should study the physiological action of the albuminoids contained in the plasma, especially of the serum-albumen. We may assume that, apart from certain *primary* reductions of the amount of water in the blood which lead to the above-described blood thickening, anhydræmia, it may *secondarily* be determined by the amount of the plasma-albuminates; an absolute reduction of the latter, or an absolute *hypalbuminosis* of the blood does not necessarily imply diminished concentration of the plasma, but only a corresponding loss of plasma water, a diminution of volume without change of concentration.

Except for the temporary variations due to food and drink, the volume of the blood is pretty constant. This constancy is rendered possible from the propelling power of the heart and elastic contractility of the vessels remaining about the same, while there is always nearly the same amount of substances capable of swelling in the plasma. Indeed, neither water nor a simple saline solution could long remain unchanged or circulate in the vessels, but more or less of it would constantly be lost by filtration through the excretory organs. Hence the plasma must contain certain substances (such as the albuminoids) which do not so much dissolve in water as swell up in it immensely, and thus retain a large amount of water in the circulation. When sufficient water is supplied from without to fill the interstices of the plasma-albumen till it is swollen to its full capacity under the existing pressure, we have the plasma in its peculiar physical condition, when it is exceedingly mobile and capable of circulating. It is entirely due to this peculiarity of the albumen that a moderate quantity of it enables a relatively large amount of water to be kept in the vessels, instead of being eliminated from the body. This will explain why the above-mentioned relation between the volume of the blood and that of the plasma-albumen must exist, why the former remains the same unless the latter changes, and why, on the other hand, an absolute hypalbuminosis usually leads to a corresponding reduction of the volume of the blood.

This rule has some exceptions, for, while the amount of plasma and albumen may be decreased, the excretion of the superfluous water may be prevented in some way, as by impaired action of the heart or kidneys. Hence, in spite of the hypalbuminosis, the volume of water is relatively too great and there is a dropsical condition of the blood (a hydræmia). As this prevention of the excretion of water also keeps in the blood a certain quantity of salts, the dropsical blood not only contains more water, but more salts, in proportion as the albumen diminishes. This peculiarity was long ago stated by *C. Schmidt,* when he showed that the loss of nine parts of albumen was accompanied by an increase of one part of salts.

Although the addition of hydræmia magnifies the symptoms of simple anæmia, the change is not sufficient to justify a separate classification; so we often speak of anæmia, when from retention of water the blood-vessels are not only not too nearly empty, but when they are abnormally filled. In such cases there is a relative hypalbuminosis, and careful examination of the blood usually shows diminution of the percentage of red corpuscles.

If, from what has been discovered by recent investigations about the blood, we attempt to define anæmia not only symptomatically, but nosologically and anatomically, we can only to a limited extent bring all cases under the same head. We must remove from this class the hydræmia due to simple retention of water; this may and often does complicate anæmia. Secondly, we must drop anhydræmia depending on lack of the proper amount of water in the blood, as it is a different condition from oligocythæmia, hypalbuminosis, etc. In general pathological histology, the latter should be termed atrophy of the blood.

It seems proper to regard the blood as a *tissue* composed of peculiar metamorphosed cells (the red corpuscles) and an albuminous basement substance (the plasma), and the whole mass of the blood as an organ of the body formed from the blood-tissue; then there will be an analogy between the states due to poverty of the blood and atrophy of other organs. As in the latter case reduction of the cellular elements (numerical atrophy or aplasia), deficient development in size (simple or proper atrophy), and different varieties of these may combine to constitute common organic atrophy; so in anæmia we may have the changes of the red corpuscles, termed oligocythæmia, oligochromæmia, etc.; and as in atrophy of organs provided with intercellular substance, this usually participates in the atrophy, so the analogue in the blood, the plasma-albumen, participates in the common hypalbuminotic poverty of the blood. The propriety of regarding the plasma-albumen as an integral component of the blood-tissue is based on the fact that most of that substance in *living* blood is necessary for the nutrition, but not directly suited for the nutrition of other tissues.

This view of the blood as an *organ,* like other parts of the body, is opposed to the old one that the blood, or at least the plasma, is not organized, but is merely a nutrient fluid. The latter view was the one held in the first edition of *v. Ziemssen's* encyclopædia. Since then the whole doctrine about the nature of plasma has changed, according to the ideas of German writers.

PATHOGENY.

Obstruction to the formation of blood, or *anhæmatosis,* and consequent hypoplastic anæmia, may be due to several causes. Development of blood-tissue requires, firstly, a sufficient supply of material from without; secondly, that this material shall be used to make *blood*. Hence, impaired assimilation of this material in the digestive tract may lead to *inanition*-anæmia, or, in spite of plenty of food and good digestion, blood formation may prove deficient because some other normal or pathological tissue uses up too much nourishment, *relative* hypoplasia: in other cases the hypoplasia is primary and absolute.

In regard to the development of plasma-albumen we can only say that, like the albumen of other organs, it is probably formed from the so-called *circulating* albumen (*Voit*) or stores of albumen in the bodily juices by intramolecular changes. If the supply of this circulating albumen from without is deficient, the regeneration of the plasma-albumen is arrested, and the same is true if other tissues take up too much of the circulating albumen.

Two derivatives of hæmoglobin, the coloring matter of bile and urine, are daily excreted in considerable quantities, which shows that a large number of red blood-corpuscles must be destroyed every twenty-four hours. If we may draw deductions from the conditions in inanition in animals experimented on by *Panum*, it would seem that the albuminates of the plasma are used up more rapidly than the red corpuscles by hunger. While the blood is undergoing active change, its physiological integrity can only be maintained by active and uninterrupted regeneration.

ÆTIOLOGY.

Experiments on starving animals show that the blood is used up or disappears in proportion to other parts of the body, acting not as a nutritive juice, but, like other histological formations of the organism, itself requiring nourishment.

Incomplete inanition has a very direct and extensive causal connection with human pathology, since from various sources it induces anæmia. Want, poverty, ignorance, malignity, or avarice may cause lack of proper nourishment and consequent lack of blood. The changes in the blood from incomplete inanition may also be better studied from the effects on animals of feeding at their regular meal times on too small quantities of their usual food; the same changes occur, as in complete starvation, but more slowly. After being starved for a time, if the animals be fed freely a few times, the blood remains more like the normal, but is impoverished in red corpuscles, thus constituting an oligocythæmia. This would seem to show that the new formation of red corpuscles requires a longer time than the regeneration of the water, plasma-albumen, and salts of the plasma; also that the physiological consumption of these latter constituents is more rapid than that of the red corpuscles.

Experiments on animals show that the danger of anæmia is greatest when the food contains too little trophic plastic material, but that restriction of fats, carbohydrates, and gelatinous substances is also injurious. For the continuance of good health and vigor of an organism it must be supplied with plenty of material for warmth and work; this end may be attained by the decomposition of large quantities of nutrient albumen, but much more easily and certainly by oxydation and breaking up of other kinds of food. Moreover, the withdrawal of fats, carbo-hydrates, and gelatin *directly* promotes anæmia, by impeding the transformation of circulating albumen into organ-albumen, and also, by hastening the breaking down of the organ-albumen.

A diet scanty in albumen may be endured longer without anæmia, if there are plenty of other substances; but where the supply of albumen is short, any reduction of other food is injurious.

Among the components of living tissue is water. Restriction of necessary drink not only causes drying of the tissues and anæmia sicca, but it interferes with the regeneration of organ-albumen. From continued thirst there is extensive parenchymatous degeneration of all the tissues, probably of the blood also, so that the effect of a thirst cure would be the same as that of a hunger cure.

When from any cause there is a considerable loss of blood, the lateral pressure in the blood-vessels is diminished, while the lymph stream is hastened and the tissue fluids flow toward the blood. Hence the blood receives more colorless lymphatic elements, water, and salts, becoming more diluted, absolutely rich in salts and leucocythæmic, while it contains relatively less plasma-albumen and normal-colored red blood-corpuscles. Other oligochromæmiæ may be referred to a more copious admixture of undeveloped red elements (poor in hæmoglobulin) with the blood, inducing symptoms like leucocytosis. As the hastening of the lymph-current, and the altered osmosis of the tissue-fluids continue some time after the arrest of the hæmorrhage, the above changes of the blood do not reach their maximum intensity at once. The regeneration of the plasma-albumen takes place more rapidly than that of the red corpuscles, hence oligocythæmia, oligochromæmia, and leucocytosis last considerably longer than hypalbuminosis.

Among the various causes of hypalbuminosis may be mentioned 1. colliquative sweats, in which the perspiration contains albumen, and the loss of water induces at the same time anæmia sicca; 2. exudative inflammations, where quantities of the contents of the vessels pass into pathological exudations, which may escape or be deposed in the body. It is very probable that the formation and consumption of blood are influenced by the central nervous system; as a result of various cerebral and spinal diseases, we sometimes have bodily disturbances which may be referred to the existing neurosis. Cases of anæmia and general marasmus from this cause are occasionally unusually malignant and may prove fatal in spite of all treatment. Recently *Schüle* has called attention to this association between organic diseases of the central nervous system (chronic meningitis, encephalitis, and myelitis) and pernicious anæmia, and has enriched this department by publishing interesting observations. Although it must be admitted that there is most probably a connection between the two, the pathological mechanism is not at present explicable and must be a subject of further study.

The blood of anæmic patients is usually of a lighter red than that of healthy persons, as is shown by examinations of specimens taken during life, as well as by post-mortem examinations. The pale hue of the blood is most marked as patients are first recovering from abundant hæmorrhages; next in those peculiar but not frequent forms of the disease combined with enlargement of the spleen and lymphatic glands, called anæmia splenica or lymphatica. It is also pale in most cases of anæmia from loss of albuminous fluids, especially excessive albuminuria and diarrhœa, and in cases due to malignant neoplasia. But in other marked cases of anæmia the blood is not peculiar in color; this is especially true in cases due to fever, while the fever lasts, whereas the paleness is said to appear during convalescence. In still other cases, especially in those complicated with anhydræmia, the blood may be unusually dark and thicker than normal.

To describe the coloring power of the blood numerically, various "hæmochromometric" methods have been proposed of late. The first attempts were by *H. Welcker* in 1854, in his publications about counting blood-cells; he described two processes for color test. One of these was to dilute one part of the blood with 1,000 parts of water, and compare the color of the mixture with that of a scale of tints made by mixing normal blood with water in certain proportions; the other was to compare the spot made by dropping the diluted blood on white paper, with a scale of blood spots made from the above solutions. Subsequently, solutions of pure hæmoglobin were used as the color-tests, and the spectroscopic peculiarities of hæmoglobin were made use of in various ways for chromometric examinations of the blood. *Vierordt's* method of determining the quantity of hæmoglobin is based on the use of the spectroscope, and the coloring power is photometrically determined; or, what is the same, the relative amount of coloring matter, according to the rate of light absorption shown by the spectrum from a regular light through a layer of 1 cm. thick, of a one-per-cent solution of the blood to be examined. Other chromometric methods have used substitutes for hæmoglobin, such as solutions of picrocarmin (*Rajewsky*) in water kept in capillary tubes; or in gelatin in hollow glass prisms, where the differing thickness of the gelatin layer corresponding to the color of the solution would be the measure. *Hayem* suggested a scale composed of strips of colored paper.

From the examination of the blood in anæmia, it seems that its coloring may be reduced to one-third or one-fifth of the normal. *Immermann* found the coloring power of the blood in one case of anæmia splenica five times less than normal, showing a reduction of the hæmoglobin from the normal 15 per cent to less than 3 per cent; a reduction to 12 or 10 per cent in mild cases of anæmia is not uncommon. In other cases of impoverished blood, the percentage of coloring matter is reduced slowly, or not at all, as in anhydræmia or acute fevers. In 20 cases of typhoid fever, *Immermann* found no increased paleness of blood during the fever, but it occurred after the fever.

In anæmic persons the volume of blood is usually reduced, sometimes very much so. Although an accurate measurement of the blood cannot be made during life, or scarcely so even after death, certain symptoms during life (diminished fullness, color, pulse, etc.) would show the reduction, as would the results of post-mortem examination. The smallest amount of blood is found in cases of acute anæmia from hæmorrhage, but the bodies of patients who have died from phthisis, cancer, and other exhausting diseases are sometimes greatly impoverished in blood. There is also great reduction in pronounced anhydræmia, or anæmica sicca, so that cholera corpses in this respect much resemble those of persons who have bled to death. On the other hand, cases occur where the diminution of volume is less marked, or, at least, is secondary to other changes in the blood, such as lack of color; this may even be true in hæmorrhagic anæmia, if we do not see the patient within a few days after the loss of blood. The contrast between the paleness of the blood and fullness of the vessels is usually great in hydræmia, due to retention of quantities of water in the blood. In these cases the vessels may seem overfilled, the blood volume being increased by the excess of water (a serous plethora). Such cases occur in acute parenchymatous nephritis, where the reduction of urine has not yet been followed by dropsy.

Quincke has described a method of measuring approximately the volume of blood, especially in excessive anæmia during life. For the success of the attempt there must be a marked difference in the richness in red corpuscles, between healthy blood and the blood of the patient. The plan is first to count by *Malassez's* method the number of red corpuscles in one cmm. of a one p. c. solution of the blood of the patient, then transfuse some healthy human blood whose blood-corpuscles have been estimated (from counting those in 1 cmm.) and after the tranfusion again count the number of red corpuscles in the blood of the patient; now knowing the amount of blood transfused, from the difference of the counts before and after transfusion, we may determine the volume of blood in the patient, by the formula $x = t. \frac{b-c}{c-a}$, in which

x=the volume of blood in the patient before transfusion;
a=the number of red corpuscles in 1 cmm. of a one-per-cent solution of his blood before transfusion;
b=the same in the blood transfused;
c=the same in the blood after transfusion;
t=the amount of blood transfused in ccm.

Of course this method would only be approximate; and it is based on the supposition that the blood transfused mingles regularly with the blood of the patient, and that the latter does not change its volume by taking up water, or reduce it by giving off water, and that no red corpuscles are destroyed during the time occupied; but it may answer in cases of marked anæmia, and to measure the results of treatment.

Coagulation of the blood of anæmic patients differs from that of normal blood, in the fibrinous clots being more scanty and loose; at least in the majority of chronic cases. In acute cases due to hæmorrhage, the blood escaping last forms hard coagula, which moreover develop very rapidly.

Chemical analyses of the blood in anæmia have been made, with the result of finding, in many cases, deviations from the normal. Especially often was there diminution of the percentage of *iron;* but this is not true in all cases of impoverished blood. Where there is anhydræmia, as in acute fevers, the percentage of iron in the blood may even be increased. About the same is relatively true of *albumen,* the percentage being usually diminished; if there be decided hydræmia, the blood appears "watery," and chemical examination shows that not only the water, but also the *salts* have increased. In anhydræmia, besides increased percentage of iron and albumen, there is an increase of the salts. In anæmia sicca there is a decided increase in the *potash* salts.

Some important changes in the blood of anæmia patients have of late been discovered by *microscopic* examination. These changes of course affect the formed elements, the red and white corpuscles, especially the former, whose relative number, color, size, and form vary from the physiological standard. The colorless corpuscles increase in some forms and cases of anæmia, as in recovery from acute cases after hemorrhages, and in some chronic cases besides true leucocythæmia.

In relative oligocythæmia the blood is proportionately poor in red corpuscles, they having decreased more decidedly than the other constituents; where this condition exists, counting the red corpuscles under the microscope shows them to be less in number than normal (5,000,000 in 1 cmm. of blood of a man, 4,500,000 in that of a woman according to *Welcker*). This reduction is not unfrequently so great that their proportion to the normal is only one-half, more rarely one-third, fourth, or even one-fifth. In anæmia sicca and that from acute fevers, the proportion of red corpuscles may be increased instead of diminished. The lack of colored elements is usually greater the paler the blood, and the reverse; but microscopical enumeration sometimes shows them to be more or less abundant than the color of the blood would have led us to suppose.

The method of counting blood-corpuscles as perfected is as follows: The principle is to measure accurately a very small amount of the blood to be examined, to dilute accurately with a certain amount (usually one hundred times its bulk) of some fluid which does not change the blood-corpuscles, and then spread it evenly on the slide. By counting the red corpuscles in a known fraction of the portion on the slide, and multiplying the result by the divisor of the fraction and this by the grade of the dilution (one hundred?) we have an approximation to the number of red corpuscles in the original blood.

Different methods merely differ in the details of performing the above acts. That of *Malassez,* which is at present the most used in Germany, is about as follows: With *Potain's* mixer or "melangeur" (Fig. 1), blood fresh from a wound on the patient is to be drawn up till it reaches *b* on the capillary tube, which is just the one hundredth part of the space *b–c* comprising the reservoir *a,* then the diluting fluid (Natr. sulphur, 5.0; glycerin, 25.0; aq. dest., 100.0) is drawn in till the fluid rises to *c*. By carefully shaking, the glass ball *e* in *d* causes an even mixture of the blood and diluent, then the part of the diluent in the tube up to *b* is pressed out and the hundred parts of mixture are forced into the capillary tube *g* on the object slide *f* (Fig. 2): then under a microscope the number of red corpuscles in one field are counted by aid of a micrometer eye-piece in different portions of equal length of the capillary tube. *Gowers'* hæmacytometer is used in England. In it the floor of the cell which holds the diluted blood is ruled in squares the tenth of a millimetre in size. The number of corpuscles in ten squares is counted and this multiplied 10,000 gives the number of corpuscles in a cubic millimetre of blood.

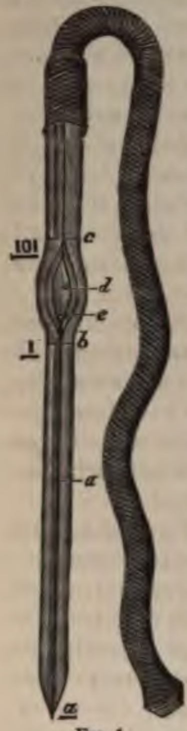


FIG. 1.

Another change of the blood recognizable by the microscope is oligochromæmia, or lack of hæmoglobin in the individual red blood-corpuscles. It has for some time been noticed in acute anæmia from hæmorrhage that the red corpuscles are not only fewer but paler. Later it has been found that this is also a common symptom in chronic anæmia. Where the lack of color is marked, it can readily be detected by the microscope; when it is less decided, the apportionment of the hæmoglobin to the red corpuscles may be determined by *Malassez's* method. By any of the hæmochromometric methods the absolute quantity of hæmoglobin in a given quantity (1 cmm.) of the blood to be examined, is determined, then the number of red corpuscles in it is counted and the former amount is divided by the latter. We thus obtain an approximate numerical expression of the hæmoglobin contained in the individual red corpuscles, which may easily be compared with the normal. In cases of severe oligæmia the proportion of hæmoglobin may fall considerably, even to less than half the normal amount. The amount of hæmoglobin in a red corpuscle of a healthy adult is about 30 billionths of one gram.

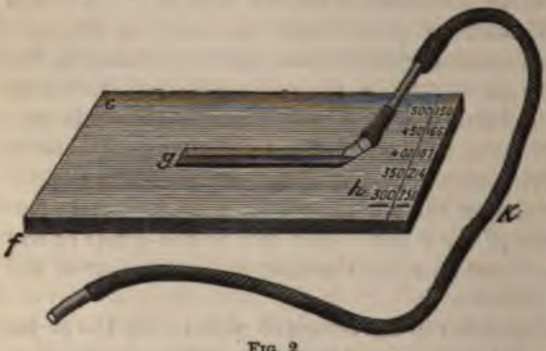


FIG. 2.

Red corpuscles vary also in size during disease; in health their diameter is not always exactly the same, but about 75 per cent of them will be the same size, having a diameter of 75 one-thousandths of a millimetre, or a little over $\frac{1}{3500}$ of an inch; even in healthy blood there will be some larger ones even of the diameter of 90 thousandths of a millimetre and others as small as 60 thousandths.

Anæmic blood shows various deviations from the above. This is not so much the case in acute anæmia from hæmorrhage as in chronic forms from any cause; in which, as soon as the anæmia becomes marked, the medium size of the red corpuscles falls from 75 to 70 or even 60 thousandths of a millimetre. The same is true in subacute anæmia from fevers, except that the medium size is usually reduced more rapidly. In true chronic forms after a time corpuscles may be much smaller than any in healthy or feverish blood, and they may not be larger than 60 or even 20 thousandths of a millimetre. These microcytes (*globules nains* of *Hayem*) seem more susceptible than normal blood-corpuscles and while being examined under the microscope lose their biconcave form, becoming flattened or perhaps spheroidal. If the microcytes in anæmic blood are very numerous, as sometimes occurs, we have the state described by *Masius* and *Vanlair* as microcythæmia. In opposition to this dwarfing, there may be giant growth of the red blood-cells in anæmia. Megalocytes (*globules géants* of *Hayem*) may attain a diameter of 120 to 140 thousandths of a millimetre, far surpassing that of large normal corpuscles; these corpuscles are less consistent, less concave and less colored than the others; they occur singly and only in severe forms of anæmia, while microcytes are found in medium grades. The accounts of these corpuscles require further corroboration and investigation, especially as to whether there is not a change in acute anæmia also of the dimensions of the red corpuscles, and whether the spheroidal form of microcytes is really artificial, as suggested by *Hayem*, or whether some of these spheroidal bodies in microcythæmia do not exist in living blood.

Sometimes there are other deformities of the red corpuscles in anæmia. In some cases of pernicious anæmia *Quincke* found besides oligocythæmia numerous microcytes and deformed red corpuscles, with hook-like processes, bottle-shaped or shaped like the incus and malleus. This condition Q. called poikilocytosis; as it is found not only in idiopathic pernicious anæmia, but in symptomatic forms (as A. splenica, A. from phthisis, cancer, etc.) and as it may be absent in the former, it seems best to regard it as not characteristic of any special form, but as an occasional occurrence in any of the forms of severe anæmia. It has not been found in the milder grades.

Nucleated red blood-corpuscles, such as occur normally in the blood of infants born at term, and in some cases of leucocythæmia, especially of the medullary form, do not seem to exist in ordinary anæmia, even when excessive or pernicious. On the contrary, in the latter cases, as well as in those of medullary pseudoleucocythæmia, nucleated red corpuscles have been repeatedly found in the medulla of the hollow bones.

Coagulation of blood is due to formation and deposit of fibrin, which seems to be a chemical union of two albuminates existing in the blood, fibrinogen and fibrinoplastic matter, the union of which two bodies is probably accomplished by aid of a ferment existing or formed in the blood. The fibrinogen probably exists in the blood, while the other two are most likely products of decomposition of the colorless corpuscles. The *amount* of the fibrin depends on that of the fibrinogen in the plasma, it being the predominating constituent. The *rapidity* of the deposit depends on the rapidity of the destruction of the leucocytes, which is the first step in the death of living blood.

The fact that the amount of fibrin which separates from the blood in anæmia is usually less than normal shows that there is less fibrinogen; while the frequency of rapid coagulation indicates increased frailty of the white blood-cells. Both peculiarities seem to show that in anæmia, especially when chronic, the physiological integrity of the blood is impaired in more than one direction.

The leucocytosis always present in acute anæmia is absolute, not merely relative; whether the same is true in chronic anæmia is a different question. In the latter case the appearance may be due to the greater proportional diminution of red corpuscles.

In some cases, numerical decrease of the red corpuscles cannot be detected by counting through the microscope, as they diminish in the same proportion as other constituents of the blood.

Oligocythæmia may be due to hypoplasia (diminished new formation) or consumption (increased destruction) or the two combined.

Oligochromæmia is apparently due to imperfect development of the blood; it is usually absent in acute fevers, where the red corpuscles are probably destroyed very rapidly, while it generally occurs when the blood is filled with young, imperfectly formed hæmocytes, or when the cytogenous apparatus has become so much impaired that perfect red blood-corpuscles cannot be formed. Oligochromæmia is found in the subsiding stage of acute anæmia after excessive loss of blood, in convalescence from severe fevers or from various chronic diseases at a period when the *number* of red corpuscles has nearly returned to the normal.

Little is known about the nature and significance of microcytes; the reduction in size of the red corpuscles in fevers is probably the first step in their destruction, and is perhaps referable to loss of water from the blood. In *chronic* anæmia also *Quincke* regards microcythæmia as due to consumption of the hæmocytes, while *Hayem* and others regard it as due to defective hæmatopoiesis.

Poikilocytosis (variation in form of the red blood-cells) is found under similar circumstances as the above, but in more pronounced cases, and involves a bad prognosis.

Tendency to dropsy in cases of anæmia is greatest when hypalbuminosis and hydræmia co-exist; this occurs typically in severe nephritis with albuminuria and diminished secretion of urine, but in these very cases there may be no dropsy. Experiments on animals, by bleeding and subsequent transfusion of salt water, have shown that hypalbuminotic hydræmia does not at once cause dropsy of the subcutaneous tissue and serous cavities, but *predisposes* to dropsy; hence, besides the dropsical dyscrasia, there is another factor of more variable, accidental, and local nature. This factor is most probably a morbid permeability of the capillary walls, although no nutritive changes have as yet been discovered in them; but very slight changes may induce unusual permeability, as is seen in exudative inflammations, where the contents of the vessels pass through apparently unchanged walls. This change in the vessels has been termed "hydropoginous" or dropsical.

Now comes the question why this change in the vessels so frequently coincides with hydræmic anæmia. Experiments have proved that the normal function of the capillaries of keeping the blood capable of circulating and allowing only a moderate quantity of its constituents to escape depends on a normal condition of their contents. For a constantly renewed contact of the wall of the vessel with fresh, healthy blood is necessary to keep it in condition to fulfil its duties to the blood, and even temporary disturbances suffice to induce an unusual permeability of the capillaries, not only for water, salts, and plasma-albumen, but for blood-corpuscles also. It is probable that a considerable impoverishment of the blood in hæmoglobin and plasma-albumen, as occurs in advanced anæmia, would have much the same effect on the capillary walls as a temporary interruption of the blood supply. Hence the permeability of the capillary walls may be due to their deficient functional restitution by the defective blood.

A further factor favorable to the dropsy of anæmia is weakened action of the heart, causing venous congestion from the effect of gravity on the motion of the blood.

One effect of anæmia is fatty degeneration due to lack of hæmoglobin and consequent deficient supply of oxygen. It has been lately observed that temporary impairment of the interchange of gases in the lungs, or deficient supply of oxygen to the tissues, is followed by an increased excretion of urea for some days.

This would seem to indicate that lack of oxygen in the blood favors the death of organ-albumen. This excessive excretion of urea from the nitrogenous products of decomposition of the organ-albumen is not balanced by non-nitrogenous products (carbonic acid and water), the latter being even relatively less than normal; hence it would seem probable that the non-nitrogenous products from the organ-albumen of the tissues in anæmia remain in position unconsumed, in the form of fat, instead of being oxydized to carbonic acid and water. While the urea is increased, the carbonic acid excreted in anæmia is diminished, as shown by the experiments of *J. Bauer*. Certain causes of anæmia, especially fever, increase the excretion of carbonates; this excess will continue while the fever lasts.

The excretion of water in anæmia as in health depends greatly on the amount of drink and on the activity of the excretory organs, skin, lungs and kidneys. When the heart's action is feeble, the lateral pressure in the arteries is reduced, and the secretion of urine less copious. Anæmia may affect the *production* of water in the body by preventing oxydation.

Increased excretion of iron in anæmia has not been observed; then what becomes of the iron from the hæmocytes that are destroyed? It would seem from recent observations of *Quincke* that the iron is deposited in some of the organs of the body, as in the liver, pancreas, kidneys, in shape of albuminate of iron.

ESSENTIAL PERNICIOUS ANÆMIA.

BIBLIOGRAPHY.

Habershon: Medical Times and Gazette, March 4th, 1876.—Broadbent: Practitioner, Jan., 1875.—J. Chadwick: Boston Med. and Surg. Journ., Jan., 1875.—W. Pepper: Amer. Journ. Med. Sci., 1875, 1877.—Pye-Smith: Virchow's Archiv, Bd. LXV.—Lebert: Archiv Gén., 1876, and Handbuch d. allg. Pathol. u. Therap., II. Auflage, 1876.—Scheby-Buch: Deutsch. Arch. f. klin. Med., Bd. XVII.—H. Quincke: Samml. klin. Vorträge, 100; 49 Versamm. d. Naturforscher u. Aerzte, 1876; Deutsch. Archiv f. kl. Med., Bd. XX.; Siderosis, Festschrift, etc., Berne, 1877; Centralbl. f. d. med. Wissenschaften, 1877.—H. Eichhorst: The same, 1876; Die prog. pernic. Anæmie, 1878, Leipzig.—E. H. Bradford: Boston Med. and Surg. Journ., 1876.—Grainger Stuart: Brit. Med. Journ., 1876.—Bradbury, do.—Lepine: Union Méd., 1876; Revue mensuelle de méd. et de chir., T. I., Nos. 1, 2.—Vallin: Gaz. hebdom., 1876.—Mignot: Union Méd., 125.—Strumpell: Archiv d. Heilkunde, 1876.—Cohnheim: Virchow's Archiv, Bd. LXVIII.—Ferrand: Bulletin de la Société des Hôpitaux de Paris, 1876.—Gowans: Glasgow Journ., T. VIII.—Bramwell: Brit. Med. Jour., 1876; and Med. Times and Gaz., 1877.—Andreas: Med. Times and Gaz., 1877.—Glynn: Lancet, 1877.—Treves, do.—Richlin: Gaz. de Paris, 1877.—Lycke: Norsk. Magazin, etc., Bd. VI.—Stricker: Charité-Annalen, Berlin, 1877.—Davy: Lancet, 1877.—Hoffmann: Dorpat Med. Zeitschrift, IV.—Rosenstein: Berl. klin. Wochen., 1877.—Nykamp, do.—H. Muller: Die progressive pernic. Anæmie, etc., Zürich, 1877.—Osler and Gardner: Centralblatt f. d. med. Wiss., 1877.—Litten: Berlin. klin. Wochenschr., 1877.—Eisenlohr: Deutsches Archiv f. klin. Med., Bd. XX.—E. Fränkel, do. Bramwell: Edinburgh Med. Journ., Vol. XXIII.—Pepper and Tyson: Virchow's Archiv, Bd. LXXI.—Pilz: Deutsch. Archiv, Bd. XXI.—Purser: Dublin Journ., LXIV.—Sörensen: Nord. Med. Ark., IX.—Wernick: Deutsch. Archiv f. klin. Med., Bd. XXI.—Bell and Osler: Transac. of the Canada Med. Assoc., Vol. I., 1877.—Wasastierna: Firmska läkares, XVIII., 1876.—Holsti, do., Vol. XIX.—Makenzie: Lancet, 1878.—Warfvinge: Hygiea, XXXIX.—Lodi: Riv. Clin., T. VIII., 1878.—Flint: New York Med. Record, 1878.—Bull: Norsk. Mag., Bd. VIII.—Haddon: Edinburgh Med. Journ., Vol. XXIV.—Rühle: D. med. Woch., IV.—Pokrowski: Petersb. Med. Woch., III.—Taylor: Guy's Hosp. Rep., XXIII.—Stephen Mackenzie: Lancet, 1878 (see Braithwaite's Retrospect, Part LXXIX.

In the first edition of v. Ziemssen's Encyclopedia, *Immermann* terms this disease "Progressive pernicious anæmia." But as cases of anæmia, due to discoverable causes, seemed to have been classed by other writers under this head, *Immermann* seems to consider it proper to give a name, limiting the class of cases to those where no ultimate cause is discoverable, and now terms it "*Essential* pernicious anæmia."

These undiscovered causes of essential anæmia seem to be unevenly distributed over the earth; in the canton Zürich, in Switzerland, the proportion of cases has been large. They have been described and commented on largely by *Lebert*, *Gusserow*, *Biermer*, and *Müller*. Possibly the causes in all cases may not be the same; some may have a specific miasmatic origin, others may be due to parasites. Pregnancy and child-bearing seem to influence the occurrence of this malady; except during this state, it is said to be no more frequent among women than among men, wherein it differs greatly from chlorosis.

One reason for seeking a specific cause for essential anæmia is the resemblance of some of the cases to general tuberculosis, or poisoning by phosphorus.

Essential anæmia is said to occur as a *secondary* disease from: 1, bad living, especially deficient nourishment; 2, repeated pregnancies and labors; 3, prolonged disturbances of digestion, particularly if accompanied by vomiting or diarrhœa; 4, losses of blood or juices of the body. However, very few of those exposed to these causes are affected with the disease; and they probably would not be more apt to induce it in Zürich, where the disease seems common, than they would in New York, where it is rare.

In a small proportion of cases of pernicious anæmia, there is some enlargement of the spleen, but not of the external lympathic glands. The clinical resemblance of this and *Addison's* disease is quite marked in some cases. *Addison* seems to have been the first to describe this disease, though it was *Biermer's* writings that attracted attention to it most. The most positive and general symptom is perhaps the change of color; next may come auscultatory changes in the heart and blood-vessels, most frequently systolic murmurs either at the apex or base.

The urine has often been found to contain indican, also creatinin and lactic acid.

Counting the blood-corpuscles shows that they may be reduced to one-tenth of the normal number. To the naked eye the blood looks watery, and the fibrinous coagulum is small and loose. The corpuscles are often smaller than normal, and of a more spherical or other unusual shape, and less inclined to form rouleaux.

Retinal hemorrhages, which are so common, on microscopical examination have sometimes been found to proceed from enlargements (capillary aneurisms) of the retinal small vessels. The yellowish or whitish centres of some of these points of hemorrhage have sometimes seemed due to accumulations of colorless corpuscles.

Although the external lymphatic glands are not affected, the mesenteric glands have frequently been found enlarged, or with red spots or irregular hyperæmias. Changes in the medulla of the bones have often been found. *Cohnheim* found the redness of the marrow not to be due to recognizable hemorrhages; the fat-cells had almost disappeared, but red and white blood-corpuscles were numerous. The red corpuscles were at least as numerous as the white, partly of normal shape (biconcave), but mostly spherical, non-nucleated, of variable size, from the normal to twice the normal size.

Various organs, as the liver, spleen, pancreas, and lungs, have been found to contain leucin and tyrosin, and some of them an abnormal amount of iron, in the shape of a finely granular albuminate deposited in the cells of the organ. The iron appears most plentifully in the peripheral portions of the liver, where it may be detected by its dark-green reaction with sulphide of ammonium, or the deep blue with ferrocyanide of potash or muriatic acid.

According to *Quincke*, the dry human liver normally contains 0.08 per cent of iron, while in two cases of pernicious anæmia he found 2 per cent in one, and 0.6 in the other. He also found an excessive proportion in diabetes mellitus. Fatty changes in the different organs are perhaps the result, not the cause of the anæmia. *Perl* has induced fatty degeneration of the heart in dogs by repeated bleedings.

In regard to the treatment of essential anæmia, the transfusion of blood, repeated if necessary, the employment of phosphorus, quinine, and more especially of arsenic (Fowler's solution, 5 to 6 drops with tartrate of iron, three times daily) have their advocates; and from the reports in regard to them, the latter, at least, deserves repeated trials. It would seem that inhalations of oxygen should do good here, as they do in chlorosis.

Notwithstanding many articles have been written on this subject of late years, there has not been a great deal that was new. Many cases have been related and suggestions made for examinations in future, and perhaps a foundation laid for better knowledge hereafter.

CORPULENCE.

BIBLIOGRAPHY.

H. Immermann: 2d edit. v. Ziemssen's Encyclopedia.—W. Mitchell: "Fat and Blood."

Corpulence depends not only on the quantity of food eaten, but still more on its variety and the frequence with which it is taken. Another important factor is the amount of muscular exercise taken. These points are taken advantage of by Dr. *Weir Mitchell* in the mode of treatment recommended in "Fat and Blood." In a very excellent review of this work (*American Journal of Medical Sciences*, January, 1878), the reviewer says: "The moisture, not less than the uniformity of the English climate, favors the acquisition of fat by lessening tissue waste. Given the same social surroundings, the same hearty appetite, the same generous supply of animal food and liquors, the same result in fat-forming could hardly occur to the American, because the dry atmosphere, and possibly the extreme variation of temperature, too much increase the rate of tissue metamorphosis. . . . It is probable that the supposed unfavorable influence of the climate of North America on the first settlers and their immediate descendants does not continue, for a change in the opposite direction seems to be slowly taking place."

Dr. *Mitchell* says, "It will be well for the physician to remember that increase of fat, to be a wholesome condition, should be accompanied by gain in quantity and quality of blood, and that, while increase of flesh after illness is desirable, and a good test of successful recovery, it should always go along with improvement in color."

In *Addison's* disease, essential pernicious anæmia and some other diseases, the blood is very deficient in hæmoglobin, and less oxygen is supplied to the organism, which favors the persistence, or even increase of fat.

Persons recovering from severe illnesses, especially fevers, such as typhoid, not unfrequently become fat during convalescence, and this change may remain permanent. This is, perhaps, because such patients have enormous appetites at a time when there is a lack of hæmoglobin, and they are taking very little exercise; in short, circumstances are temporarily most favorable for production of fat, the supply of lipogenous material being large, and oxygen being deficient in the blood. The corpulence thus developed is the *anæmic* form, while the *plethoric* form is due to excessive supply of lipogenous material in such quantity that the normal amount of oxygen in the blood cannot use it up. There are cases where these conditions appear together.

SCROFULOSIS.

BIBLIOGRAPHY.

Birch-Hirschfeld: 2d edit. v. Ziemssen's Encyclopedia.—Buhl: Lungenentzündung, etc., Munich, 1872.—Villemin: Etudes sur la tuberculose, etc., Paris, 1868.—Waldenberg: "Die Tuberculose, etc.," Berlin, 1869.—Klebs: Virchow's Archiv, Bd. XLIV.—E. Ziegler: Experimentelle Untersuchungen über die Herkunft der Tuberkel-Elemente, Würzburg, 1875.—C. Friedländer: Volkmann's Samml. klin. Vorträge No. 64; and Virchow's Archiv, LX.; 50 Versam. Deutscher Naturf. u. Aerzte in München.—Cohnheim: Vorles. über Pathologie.—J. Rabl: Med. Jahrb. d. Wiener Aerzte, 1876.—Robert Bell: Lancet, 1877.—J. G. S. Coghill: Practitioner, 1877.—F. Betz: Memorabil., XXII., 1877.

Recently *Buhl* has reiterated his belief "that the cheesy masses must develop and nourish the specific virus of scrofula from themselves," at the same time saying that the *bacteria* found in them must have some effect on the decomposition taking place in them, so that we must acknowledge the action of these organisms in developing the infecting matter.

The frequent occurrence of tuberculosis in scrofulous subjects, where cheesy products are most common, furnishes a striking argument for the correctness of *Buhl's* theory of infection, as do the experiments of *Villemin*. But in this field of experiment the results of experiments as well as their explanation varied. At present there are two principal views. According to one, as above mentioned, the retrogressive metamorphosis, especially cheesy degeneration of inflammatory products, is the starting point of tuberculosis. The primary affection, the inflammation whose products become caseous, requires no specific cause. The becoming caseous depends on constitutional predisposition, such as exists in certain animals, as rabbits and guinea pigs. Some of the supporters of this theory consider the caseous material as a necessary step between inflammation and tuberculosis, while others believe in the development of the irritant causing the tubercle at the seat of primary inflammation without any caseous point. The nature of this irritant is regarded by *Waldenburg* as mechanical, by *Buhl* and most others as a specific virus formed by caseous degeneration in the body. This theory is supported by numerous experiments of *Waldenburg, Cohnheim, Fox, Sanderson*, and others, but has always had opponents, among them *Villemin* and *Klebs*. The latter claims that tuberculosis is excited by a specific virus which is developed outside of the body, and that caseous substances are only infectious when mixed with this specific agent. This view would imply a specific character in the primary inflammations from which tuberculosis originates, and this is an important point for deciding the position of scrofulous conditions.

This theory, which represents tuberculosis as being excited by a specific infection, as syphilis is, has become more popular lately, even some of its former opponents (*Cohnheim, Fränkel*) having been converted to it, although it is not universally believed that low organisms are the bearers of the tuberculous infection. This revolution in favor of the specific infection theory of tuberculosis is especially due to a sharper criticism of the experiments and the advance in histological knowledge of tubercle. This advance has changed the boundaries between tuberculosis and inflammation as laid down by *Virchow;* making it probable that the most important anatomical products of scrofula are to be reckoned as tuberculous, careful histological study having proved them to contain well characterized tubercle. *Buhl* claims that in almost any extensive embryonal connective-tissue neoplasia tubercle may be found. If such experiences seemed to indicate a very close connection between tubercle and connective-tissue neoplasia (granulations), while such a general occurrence of local tuberculosis endangered the specific infection theory, new arguments were brought up calculated to give a view of the genesis of tubercle different from the old views. Among these were especially the investigations of *Ziegler*, quoted in Vol. V. of v. Ziemssen's Encyclopedia, by *Rindfleisch*, and more recently (1878) of *Baumgarten*, on the development of giant cells about foreign bodies.

Careful revision of the results of inoculation of animals showed that many of the results did not at all correspond to human tubercle, the histological characters of the latter being absent in them. *Friedländer* even doubted if true tuberculosis was ever induced in animals experimentally, and thought the pathogeny of tubercle was no better known than that of carcinoma.

Further discussion of the histology of tubercle led to the belief that the giant cells, whose occurrence was formerly regarded as so important, were neither a specific sign nor constantly present in tuberculous neoplasia. They are not found in the typical miliary tuberculosis of the cerebral meninges, but are found in the most varied processes.

So there was a return to *Virchow's* definition of tubercle as a submiliary neoplasia composed of closely packed round-cells, without vessels, characterized by its tendency to central caseous degeneration, and its inclination to local dissemination and generalization.

If Friedländer's position be considered as one-sided, it must still be acknowledged that in many cases simple inflammatory processes (especially lobular pneumonia) were rashly presented as proofs of an experimental induction of tuberculosis. But there remained numerous cases where this error could not be asserted, and recently careful experiments have been reported as proving the transfer of tuberculosis to animals.

Cases reported before the Munich convention of naturalists attracted great attention. (Amtl. Bericht der 50. Vers. deutscher Naturf. u. Aerzte in München, S. 268–282.) In some of the cases, infection was induced by causing dogs (which are not disposed to tuberculosis) to inhale phthisical sputa; there could be little doubt about the nature of the nodules induced in the animals. (There were giant-celled tubercles, also those consisting of closely packed round-cells, so-called lymphoid tubercles.) *Klebs* also referred to experiments (infection of animals by minute organisms from tuberculous foci by the so-called fractional cultivation) which seemed to prove that tuberculosis might be induced by certain organisms (monas tuberculosum), which could be bred outside of the body. From some new experiments that he has made, *Cohnheim* has relinquished his former views of the non-specific origin of tuberculosis. Hence, if this theory has not obtained universal credence, it has at least gained ground from later experiments; its supporters meet the objection made to it, that tubercle nodules are found in processes whose infectious nature is improbable, by saying that these giant-celled tubercles, as well as those experimentally induced by Ziegler, are to be distinguished from true tubercles, whose pathognomonic quality is their "peculiar mode of transfer and extension in the body." (*Klebs* and *Ziegler* in Tagebl. der 50. Vers. d. Naturf. u. Aerzte, S. 281, and *Cohnheim*, Vorles. über Pathologie, 1. S. 615.)

Hence it is necessary for the scrofulous process, where histologically well characterized tubercle has been discovered, to find whether it is *genuine tuberculosis;* whether, for instance, fungous inflammation of joints, where *Köster* has found miliary tubercle developed in granulation tissue, can be distinguished from what *Cohnheim* calls genuine tuberculosis. *Rail* asserts that scrofulous changes are characterized by the development of peculiar granulation tissue, not by the occurrence of tubercles.

Our opinion in regard to scrofulosis will vary with the theory we hold about tuberculosis, the two states being closely related.

If, as *Cohnheim* has experimentally shown, the inflammatory exudation depends on an alteration of the walls of the vessels, and the composition of the exudation is proportional to this alteration, if the arrest of the exudation and its reabsorption is determined by the restoration of the integrity of the wall of the vessel, if the character of the neoplasia depends on the circulation which determines the nutrition, then it would seem that the abnormal susceptibility to inflammation observed in scrofulous patients, and the tendency to chronicity of these inflammations, their slight inclination to resolution, etc., would be due to a defective condition of the walls of the vessels, and more distantly to an abnormal condition of the blood. Although the above hypothesis seems to have some foundation, it does not add much to our knowledge of scrofulosis, as we do not understand the nature and causes of these alterations of the vessels. *Moretti* suggested that scrofulous diseases were induced by low vegetable organisms. *Hüter* referred them to dilatation of the vessels by the large supply of nutritive fluids passed through them in youth. As these vessels extend to the superficial layers of skin and mucous membrane, they lose their firmness which would protect them from irritating organisms in the air, and in this way scrofulous disease of the skin and mucous membrane would develop and extend through the lymphatic vessels to the glands.

As to the reasons for believing in a parasitic origin of scrofula or for its spread by a specific, unknown virus which is formed outside of the body, we are aware that they are based on no certain anatomical knowledge, nor has it been proved that low organisms constantly occur in scrofulous foci, whose specific nature is shown by special morphological peculiarities or by their activity in experiments on infection.

If it be urged that other pathological processes, whose infectious origin is undoubted, have not been proved to have a peculiar character differing from non-specific inflammations or any connection with the occurrence of low organisms, the question arises whether there is anything about scrofula which renders its specific infectious origin probable. There are no epidemics or endemics of scrofula as of other infectious diseases, nor does it spread by a fixed contagion as syphilis does. Several children in the same family may suffer from scrofulous diseases, but these do not usually occur at the same time; they are attacked independently, often at a certain age, sometimes only part of the children are affected (the males or females), while the others escape. When in a community, such as a foundling asylum, scrofula is common, it does not appear due to the crowding or to spreading from certain infecting points, but of children under the same circumstances only part will be affected, and these at intervals, as the disease preferably begins at a certain age; and on investigation we may find that most of the patients had shown a predisposition to the disease before entering the institution, and had scrofulous parents.

So we see that some of the factors from which we usually determine the infectious nature of a disease are wanting. The only one present is the fact that in the body of the patient the disease spreads like an infectious disease, as from the skin to the lymphatic glands, thence to the bones, or it changes from mild to severe forms. But this is hardly sufficient to base the hypothesis firmly, as in other cases (especially in certain tumors), where we find no infection from without as a cause of the pathological processes, disease spreads in such a way as to lead us to infer that the body is acted on by an infection in itself.

Remembering the relations of tuberculosis to scrofulosis, if we consider the former as induced by a specific infection formed outside of the body, we have only to choose whether we shall refer all scrofulous diseases to the action of the same matter, or, as *Bollinger* has done, consider that there is an infectious (tuberculous) and a non-infectious form of scrofula.

Attempts have been made to distinguish locally circumscribed tubercle from true tuberculosis, measuring the latter more by its course than by its histological characters. It is difficult to decide which scrofulous processes are tuberculous. In every extensive scrofulous granulation tissue, tubercle may be found, but the subsequent course varies. At one time, after a rich development of tubercles in granulation tissue of a scrofulous local disease, without much constitutional reaction or extension to other organs, the disease heals, and perhaps after months or years begins again at some new point; in another case, the local affection will be accompanied by disease of the next lymphatic glands, and this by development of tubercle, which may remain locally circumscribed by the scrofulous adenitis resolving or opening externally; but the disease may advance from one gland to another, and a general tuberculosis may be the result.

Lastly we may mention those cases where, without general disturbance or fever, one or more lymphatic glands slowly enlarge and become caseous; such cases are often found on autopsies of children. In such glands are found tubercle nodules, which are not found in other organs; in other cases a general acute miliary tuberculosis develops from foci apparently entirely similar.

We need not study more closely the different combinations of scrofulous diseases with each other or their relation to local or general, chronic or acute tuberculosis, to justify us in dividing tuberculosis into spurious or local, and true or general tuberculosis. In scrofulosis, the local development of tubercle is most intimately associated with inflammatory neoplasia, the proliferating granulation tissue being the seat of tubercle; and the non-vascular tissue characteristic of tubercle develops wherever the advance of granulation tissue to permanent tissue is retarded by defective nutrition. The circulation in the scrofulous foci is poor, the veins are dilated and overfilled with blood, while the lymphatics are obstructed. If we thus explain the frequent local development of tubercles in scrofulous foci without having to ascribe it to a specific infection, it is more difficult to explain why this should spread from one focus and not from another.

It is very probable that the extension is due to substances taken up by the blood from the first foci, and the tendency to spread may depend on the quantity of seed and on the conditions for its absorption, and finally on the susceptibility of the tissues secondarily attacked. On the other hand, it may be said that the local development of tubercle shows a disturbance of the normal development of the inflammatory neoplasia, but not the presence of a material infectious to the whole body. This may perhaps not occur till there has been some decomposition due to regressive metamorphosis (such as caseous degeneration); this result of decomposition having the peculiarity, when taken into the blood and carried to the connective tissue of different organs, of inducing those non-vascular neoplasia with tendency to disintegration that we call tubercle. Under the latter hypothesis we might suspect that some ferment from without aided in developing the tubercle, and perhaps bacteria might be the ferment.

If we suppose that the frequent connection of scrofulosis and tuberculosis is due to the anomalies causing the former, giving inflammations a tendency to incomplete development of tissue and regressive metamorphosis, we would not mean to assert that tuberculosis develops exclusively from a scrofulous constitution. This would not agree with the experience that often persons become tuberculous who never had any symptoms of scrofula. Apart from the direct transfer of tuberculosis, it is easy to understand that certain local states may induce an inflammation to become tuberculous and that weakness of the constitution from other causes may do the same.

In ten cases of lymphatic glands removed from the necks of scrofulous patients, *Birch-Hirschfeld* found tubercles nine times; there was also hyperplasia. He contradicts *Rabl's* assertion, "No one can find a fresh non-caseous tubercle in the tissue of a normal, hyperplastic or scrofulous gland," and says there is no better place to study well-characterized tubercle.

According to *Rabl*, most scrofulous abscesses, except cold abscesses, which occur in subcutaneous and muscular connective tissue, start from a granulation tissue, containing all forms of connective tissue, but especially embryonal; among those elements, some, by taking up plenty of protoplasm or by uniting, attain the size and form of giant-cells. The usual fate of this scrofulous granulation tissue is caseous degeneration, starting from the centre and going on to softening; at the same time there is proliferation of this tissue at its periphery. If subcutaneous, the skin is infiltrated, perforated, and the result is a scrofulous ulcer. These abscesses contain mostly softened caseous masses with some pus. What has been described as their pyogenic membrane, R. says is the remains of the scrofulous granulation tissue.

If the proliferation be located deep in the muscles, by its peripheral increase it may attain large size, and on softening cause large cold abscesses. The shape of the abscess varies with its seat; in the subcutaneous tissue it is spheroidal, in long muscles ellipsoidal, in the abdominal walls spindle-shaped.

The abnormal reaction of scrofulous patients to inflammatory irritation is shown by the occurrence of frost-bite; and *Cohnheim* has shown that such patients have a certain weakness of the vascular apparatus. In children's asylums, where the slighter ailments are always brought to the physicians, it is found that the patients suffering from perniones are those of torpid habit and with other signs of scrofula. These inflammations do not come in cold weather, but when this is followed by warm weather. Apparently the cutaneous vessels suffered the alteration leading to exudation from the cold, but the inflammation did not appear till the circulation was fully restored by the warm weather. This forms a perfect analogy with *Cohnheim's* experiments, where after temporarily arresting the circulation in certain parts, he observed the inflammatory symptoms begin with its restoration.

Cutaneous eruptions in scrofulous patients are most common on the head. In 116 cases of scrofulous eruptions reported by *Lebert*, 91 were on the head and face. Another favorite seat is about the wrist. Scrofulous eczema is characterized by free exudation (thick crusts and great swelling of the skin), great obstinacy and tendency to relapse. It is most apt to occur at the time of the first dentition, while the deeper ulcerating forms of cutaneous scrofulides usually belong to later years of childhood.

When scrofulous inflammations of the mucous membrane have existed long, they tend to hyperplastic proliferation of the mucous and submucous tissue, and especially to hyperplasia of the glands.

The lymphatic glands are most frequently attacked in connection with diseases of the skin and mucous membrane which supply them with lymph, but no considerable irritation is necessary to induce marked swelling; the glandular disease soon acquiring a certain independence and progressing even if the original affection subside. This forms a point of difference between scrofulous and simple reactive irritation, the latter subsiding with the cessation of the cause. Sometimes groups of lymphatic glands become diseased without any apparent peripheral irritation.

Scrofulous abscesses have about the same course as cold abscesses. They come without much local or general reaction, but when in the intermuscular connective tissue they impair the action of the affected muscles and cause a feeling of painful tension. In the subcutaneous tissue, where the abscesses are generally small and open early, the symptoms are usually slight. The part affected is somewhat swollen and firm, it gradually softens and fluctuates; if just under the skin, this reddens or becomes bluish-red and is thinned. In spite of fluctuation, incision may let out little matter, but on pressure a spongy granulation tissue projects from the opening. Subcutaneous as well as intermuscular inflammations not unfrequently resolve. The abscesses which open, readily form chronic or spreading ulcers and may be followed by all the results that can accompany suppuration communicating with the air.

According to *Rabl*, diseases of the bone occur in fifty per cent of scrofulous patients; this percentage is probably based on cases seen in hospital and is much too high.

The bones most frequently attacked are those of the ankle, lower epiphyses of the femur, the vertebræ, and finally the fingers and toes.

TREATMENT.

In choosing the variety of exercise for scrofulous patients, we should remember the liability of the bones to be attacked by scrofulous inflammation from injuries apparently insignificant. In some cases, on this account, the Swedish movement cure or massage may be tried.

In the treatment of children with scrofulous predisposition by a *hardening* system, they should have as much fresh air as possible; by this it is not meant that they should be made to suffer from cold either by being insufficiently clad or by being washed in too cold water. Air baths, *i. e.*, exposure of the body to the air, and rubbing well with a rough towel may be tried once or twice daily; frequent washing with warm water (not too freely used) is preferable to cold affusions or baths. Many feeble children seem markedly injured by the abstraction of heat due even to baths of moderate temperature.

Dr. *Robert Bell* and Dr. *J. G. S. Coghill* recommend chloride of calcium for scrofula and tuberculosis, especially for the disturbances of digestion accompanying the former. This is a revival of *Fourcroy's* recommendation of about a century since. It is said that the chloride may be used with advantage in some of the cases where iodine is contraindicated by bad general condition and disturbed digestion; while using it, the body may be anointed morning and evening with olive oil and attention paid to the diet. Give plenty of milk and but little starchy or saccharine foods.

Of course, in cases where there is a suspicion of syphilis, iodide of potassium should be tried.

In ordering sea-baths, we may expect most benefit from the sea-air, and often warm salt baths will do more good than ocean bathing. Sea-bathing is especially beneficial for the torpid cases of scrofula, but it also does good for erethitic cases if they are not inclined to catarrhal diseases, especially of the respiratory organs. The same points are to be borne in mind when ordering mountain air; feeble individuals disposed to catarrh should be sent to places protected from rough weather and raw breezes.

In scrofulous photophobia Dr. *F. Betz* recommends the internal use of opium at bed-time, and cold compresses during the day; also application by the atomizer for three to five minutes, of a solution of extract of opium, one part to one hundred of water, the atomizer being held eight to twelve inches from the eye, while the surgeon holds the lids apart. Then he lets the patient stay in the room half an hour.

MALIGNANT LYMPHOMA.

BIBLIOGRAPHY.

WINIWARTER: v. Langenbeck's Archiv, Bd. XVIII, and Med. Jahrb. d. Wien. Ges. d. Aerzte, 1877.—BOECKLE: Gaz. méd. de Strasbourg, 1876.—MOSLER: Berlin. klin. Wochenschrift, 1876.—BIRCH-HIRSCHFELD: 2d edition of v. Ziemssen's Encyclopedia.

Winiwarter says that in malignant lymphoma he has never been able to prove an increase of the white blood-corpuscles, but on the contrary has often found them lessened in number; the red corpuscles were paler and less inclined to form rouleaux; the patients were not particularly anæmic.

In some few cases of constitutional syphilis, there is such extensive gummous disease of the lymphatic glands that it may be mistaken for malignant lymphoma. In one case the diagnosis was not confirmed till after death; enlarged glands in the neck, mediastinum, and abdomen had cheesy deposits such as are peculiar to old gummous foci.

The inefficacy of operative treatment in some of these cases is shown by a case reported by *Boeckel*. Seventy-five lymphatic glands between the angle of the jaw and the clavicle were removed from a boy æt. 3½ years, the wound healed in a fortnight; after three weeks, by another operation forty-five glands were removed; after five weeks, a recurrence called for another operation; after seven weeks another; in seven months the space from which the glands had been removed was filled with new tumors, new groups of glands in other parts of the body were attacked, and there was a decided cachexia.

The only treatment, thus far proposed, which seems promising is that by arsenic. *Birch-Hirschfeld* reports two cases where he has tried it; one was the case of a landlord 38 years old, of feeble constitution, in whom during a year large tumors of the cervical and maxillary glands developed. In three months, under the arsenic treatment, the tumors disappeared and he remained well for three months, increasing in weight and healthy appearance; then came a rapidly progressing pulmonary phthisis which proved fatal in six months, but the glands did not enlarge again. The second case was in a strong man, aged 42 years, who had a lymphomatous tumor as large as two fists in the right axilla, while there was compression of the trachea, apparently due to hyperplasia of the tracheal and bronchial glands; the patient became emaciated and his health impaired. After six weeks of the arsenic treatment, the respiration became normal, the axillary tumor was reduced to the size of a walnut, the general health improved, and after six months there was no sign of a relapse.

Winiwarter and *Mosler* speak highly of the treatment by arsenic. Fowler's solution is combined with an equal part of tinc. ferri muriat. or liq. ferri dialysat. and five drops of this mixture given morning and evening in a teaspoonful of claret wine after eating. This dose may be increased morning and evening by one drop every second or third day, till some symptoms of arsenical poisoning appear; these usually come when the patient reaches a daily dose of twenty-five or thirty drops of the Fowler's solution (burning in the throat, eructation, vomiting, diarrhœa), and then the dose should be gradually decreased. This course will occupy two or three months.

It is advised to combine this internal treatment with injections into the glands of pure Fowler's solution, making two or three injections into different glands daily—about two minims to each injection. If the tumors become swollen and painful after the injections, these should be discontinued and moist warmth applied. Care should be taken to introduce the canula of the syringe into the gland tissue.

Children are said to be very tolerant of this treatment. Pains in the glands usually begin one or two hours after the injection.

A very annoying symptom is sleeplessness, for which bromide of potash is the most useful remedy; there may also be great restlessness and depression. In three patients *Winiwarter* saw herpes zoster.

One interesting symptom of the arsenical treatment of lymphoma is the fever. It is not a constant symptom, and does not occur if the glands are not reduced in size, so it is probably induced by reabsorption of the constituents of the tumors. The fever may occur in one of two forms, as *continued* with morning and evening remissions, or *intermittent* with intervals of freedom; there is often an initial chill and a hot and sweating stage. This fever *Winiwarter* has only seen in patients treated by parenchymatous injections, the attack usually beginning an hour after the injection. The tumor visibly diminished during each attack of fever. This diminution generally took place without suppuration, and caseous degeneration was never seen; hence *Winiwarter* assumes that there was a chemical solution and an absorption of the lymphoid elements, the phlogogenous action of which caused the fever; and that the increased transformation of albumen caused by the fever also tended to reduce the tumors.

The above treatment may not be very certain, but it looks more hopeful than any hitherto proposed, and must abide the test of time. *Winiwarter* who has seen many cases at *Billroth's* clinic and in his own practice, strongly recommends early extirpation of the glands first affected, and then resorting to the arsenic treatment.

IDIOPATHIC ADENITIS.

BIBLIOGRAPHY.

Birch-Hirschfeld: 2d edition, v. Ziemssen's Encyclopedia.—H. **Larry** and A. **Follet**: Gaz. méd. de Paris, 1844.—A. **Bertherand**: Traité des Adenites idiopathiques, Paris, 1852.

Lymphatic glands seem sometimes to inflame spontaneously, though on careful examination we may often find some peripheral irritation as the cause. Chronic enlargement of the glands at the angle of the jaw and in the neck may arise from caries of teeth or from nasal catarrh; the glands of the axilla may enlarge from injuries to the fingers, punctures with unclean instruments, etc. In the groin it may be induced by corns, nails in the shoes, etc. In such cases the swelling and pain are usually moderate.

What seems to be truly an idiopathic lymphadenitis is most frequent in young persons in the cervical glands. A feeling of tension is followed by rapid swelling, which may spread to the adjacent cellular tissue. A whole group of glands may be attacked, causing a nodulated swelling, which may involve the whole side of the neck. The skin is reddened and there may be great pain, and impaired motion. From the first there is usually fever and often chills. The subsequent course varies; the inflammation may resolve, but more frequently terminates in abscess. If pus forms and is evacuated spontaneously, it is found to be ordinary pus, differing from the matter of softened glands that have become cheesy. If numerous and deep-seated glands suppurate and are not opened artificially, the pus burrows, forming abscesses in the neck which may open into the mediastinum.

Sometimes after the acute inflammation has run its course, there is left a chronic hyperplasia, and caseous transformation occurs in what had been an idiopathic adenitis; then the disease may run on for months. This hyperplasia may extend to other groups of glands and develop into malignant lymphoma.

Simple adenitis may begin as a subacute or chronic affection, the glandular swelling coming on insidiously and with little pain or fever. Moderately large firm tumors, little inclined to soften or suppurate, develop. Perhaps we should not call these cases of idiopathic adenitis unless the disease remains limited to one gland or group of glands. Many of them probably belong to granular lymphomata, whose tuberculous nature has been recognized by *Schüppel*. In other cases, especially where numerous glands are affected, we cannot avoid the suspicion of syphilis, and may have to resort to trial by treatment, where the disease is denied. Many of the so-called idiopathic cases undoubtedly belong to progressive lymphoma.

It is probable that this disease is due to some infectious irritant brought by the lymph from the periphery. Patients often claim that it is induced by catching cold, straining, contusions. It is often called rheumatic bubo. It seems sometimes to be induced by hard marches. Some French authors have called attention to the frequency of non-scrofulous adenitis, especially of the cervical glands, among soldiers. One out of every sixteen French soldiers in the surgical wards had inflammation of the cervical glands; the proportion to the entire garrison being one to one hundred and thirty-two.

When the inflammation goes on to suppuration, it should be treated according to surgical rules; when chronic, moist warmth should be applied and iodine mineral waters, such as those of Adelheid and Thasillo, or iodide of potash should be given. In very obstinate cases, good results may sometimes be obtained from arsenic treatment as in progressive lymphoma.

DIABETES MELLITUS.—DIABETES INSIPIDUS.

BY

HENRY N. HEINEMAN, M.D.,

OF NEW YORK,

VISITING PHYSICIAN TO MT. SINAI HOSPITAL AND TO OUT-PATIENT DEPARTMENT NEW YORK HOSPITAL;
ASSISTANT PATHOLOGIST TO ROOSEVELT HOSPITAL.

DIABETES MELLITUS.

BIBLIOGRAPHY.

Abeles u. Hofmann: Ein Fall von simulirtem D. m. Wien. med. Presse, 1876, Nos. 47 u. 48.—Abeles, M.: *a.* Der physiol. Zuckergehalt des Blutes. Med. Jahrb., Wien, III., 1875, p. 269; *b.* Zur Lehre der sacchar. Fermenten im Organismus. *Ibid.*, 1876, p. 225; *c.* Zur Kenntniss d. Glykogens, *Ibid.*, 1877, p. 551. —Adamkiewicz, A.: *b.* Ammoniak im Gesunden u. Diabetik. u. die Quelle des Zuckers. Arch. f. Anat. u. Phys., Leipzig, 1879, p. 160; *a.* Verhandl. der Phys. Ges. zu Berlin, 1878, No. 17.—Bouchardat: De la Glycosurie ou Diabète sucré. Paris, 1875.—Blau, L.: Ueber D. m. et insip. (Uebersicht). *Schmidt's* Jahrb., CLXVIII., CLXXIII., CLXXV., 1877.—Bussard: Note sur le Diabète. Gaz. méd. de Paris, 1876, No. 31.—Benson: Brit. Med. Journ., 1875, p. 522.—v. Brincken: Natron Salicyls. bei einem geheilt. Fall. D. m. D. med. Wochens., 1877, No. 39. —Bohn: Acuter D. m. mit sogenannt. diab. Coma endigend. Centralztg. f. Kindheilk., 1877, No. 6.—Bohm, R., and Hoffmann, F. A.: Zur Kenntniss des Kohlhydratstoffwechsels. Arch. f. exp. Path. u. Pharm., VIII., pp. 271 u. 489. —Bernard, A.: Leçons sur le Diabète et la Glycogenèse animale. Paris, 1877.—Brunton, T. L.: Diab., Syst. Med. (Reynolds). London, 1879, V. 427–432.—Cantani: Ueber den Diabet. Blutzucker. Molesschott's Untersuch. zur Naturlehre, 1875, XI., p. 443; Patologia e Terapia del Ricambio Materiale, I. Milano, 1875.—The Same: Der Diab. mel. übersetzt von S. Hahn. Berlin, 1877.—Czapek, F.: *a.* Beitrag zur Bestimmung des D. Harnes im polarisirten Licht. Allg. Wien. med. Ztg., 1876, No. 31–33; *b.* Eine seltene Form von D. m. Prag. med. Woch., 1876, Nos. 13, 14; *c.* Ueber den relativen Werth des Phosphors u. s. w. Deuts. Ztschr. f. pract. Med., 1876, Nr. 50; *d.* Stoffwechsel während des Gebrauchs Carlsbader Wasser. Prag. med. Woch., 1878, No. 14.—Cyr, J.: Etiologie et Prognostic de la Glycosurie et du Diabète. Paris, 1878; also Br. Med. Jour., 1878, I., p. 827.—Donkin: On the Relations between D. and Food, etc. Lancet, 1875, II., No. XXV., pp. 347, 879.—Dickinson, W. H.: Changes in the Nervous System in D. Lancet, 1878, 1, 117, 160.—De Giovanni: Communicaz. intorno al D. suc. Annal. univ. diek., etc., 1876. Virchow-Hirsch's Jahresbericht, 1876, II., p. 269. —Diabetes: (15 cases) St. George's Hosp. Repts., 1877–78. London, 1879.—Ebstein, W.: Zur Therapie des D. m. insbesondere Natr. salicyl. Berl. klin. Wochenschr., 1876, pp. 337, 501.—Foster, B.: Diab. Coma, Acetonæmia. Brit. Med. Jour., 1878, Jan. 19th.—Forster, J.: Ueber die Abstammung des Glykogens im Thierkörper. Sitzsb. d. Bayr. Akad., 1876, p. 138.—Fürbringer, P.: *a.* Oxalurie u. Oxaloptyse in D. m. u. s. w. D. Arch. f. klin. Med., XVI., p. 499; *b.* Behandlung der D. m. u. s. w. *Ibid.*, XXI., 469.—Frerichs, Fr. Th.: Ein Paar Fälle von D. m. Charité-Ann., II., 1875, p. 151.—Fleischer, R.: Beiträg. zur Chemie des diab. Harns. Deut. med. Wochenschr. Berlin, 1879, V., 218–220. —Flagge, C. Hilton: Observations on D. Guy's Hosp. Rep., 1875, 3's, XX., 174.—Gee, S.: A Contribution to the History of Polydipsia. St. Barthol. Hosp. Rep., 1877, p. 79.—Gorup-Besanez: Zoochem. Analyse, p. 131. Braunschweig, 1872.—Hicks, J. Braxton: Eczema and D. M. London Lancet, 1877, 1, 456.—Hardy: *a.* D. M. Gaz. d. hôp., May 22d, 1877; Med. T. and G., July 28th, 1877;

b. Diabète sucré. Gaz. d. hôp., Par., 1879, III., 1057, 1089.—Haas: Centralbl. f. d. med. Wissensch., 1876, p. 149.—Johannowsky: Arch. f. Gynäkologie, XII., p. 448.—Külz, E.: *a.* Kann, etc., die Zuckerausfuhr durch Albuminaten gesteigert werden? Arch. f. exp. Path. u. s. w., 1876, VI., p. 140; *b.* Ueber den Blutzucker. *Ibidem*, p. 143; *c.* Beiträge zur Kenntniss des Inosits. Sitzungsber. der Marburg. Naturw. Ges., 1876, No. 4; *d.* D. m. in Gerhardt's Handb. der Kinderkrankh., III. Tübingen, 1878; *e.* Zur Kenntniss des Leberglykogens. Pflüger's Arch. f. Phys., 1876, XIII., p. 267; *f.* Experiment. über D. Deut. Ztschr. f. pract. Med., 1876, No. 23.—Kussner: Berl. klin. Wochen., 1876, No. 20.—Lecorché, O. M: Traité du Diabète. Paris, 1877.—Lancereaux, Lepierre: Coup d'œil retrospectif sur les Diabètes. Union méd., Par., 1880, 30, XXIX., 161, 205.—Leber, Th.: Augenleiden bei Diabetikern u. s. w. v. Graefe's Archiv f. Ophthal., 1875, XXI., Abt. 3.—London, B: Beiträge zur Therapie des D. m. Med. chir. Centralbl., 1876, No. 20.—Lugagne, C.: *a.* Des lésions du pancréas dans le diabète. Vichy méd., 1879, 11, 438, 449; *b.* Du régime chez les diabétiques. Vichy méd., 1878-79.—Loeb, M.: Ein nach sechstägiger Krankheitsdauer tödtlich verlaufender Fall. Entstehung durch eine Gallensteinkolik. D. Arch. f. klin. Med., Leipzig, 1879, XXIV., 343-350.—London Lancet, 1878, VI., p. 689.—Markownikoff: Aceton im Harn der Diab. Ann. der Chemie u. Pharm., 1877, CLXXXII., p. 362.—v. Mehring: *a.* Zur Glykogenbild. in der Leber. Pflüger's Archiv, XIV., p. 274; *b.* Ueber die Abzugswege des Zuckers aus der Darmhöhle. Archiv f. Anat. und Physiol., 1879, p. 379.—Meyer, Jaques: Beitrag zur Lehre von der Glykogenbildung in der Leber. Pflüger's Archiv, 1878, XVIII., p. 164.—Oppolzer: Uebersicht. Darstellung der mir zur Behandl. gekom. Fälle von D. m. Heller's Archiv f. Phys. u. Path. Chemie, N. F., 1852, p. 401.—Pick: Hautkrankheiten bei D. m. D. Zeitschr. f. p. Med., 1876, No. 32.—Pary, J. W.: *a.* On the Production of Glycosuria by the Effect of Oxygenated Blood on the Liver. Brit. Med. Jour., 1875, Vol. II.; *b.* Physiology of Sugar in Relation to the Blood. The Med. Examiner, 1875, July, ff.; *c.* Croonian Lectures on D. Lancet (London), 1878; *d.* Case of D. Associated with Temporary Mania. Med. T. and G., London, 1879.—Quincke, H.: Ueber Coma Diabeticum. Berl. klin. Wochenschr., 1880, No. 1.—Ryba u. Plumert: Behandl. des D. m. mit salicyls. Natr. Prag. med. Wochens., 1877, Nos., 19-21.—Redon: Virchow-Hirsch's Jahresber., 1876, I., p. 264; Du D. M. chez l'enfant. Compt. rend. Soc. de biol. 1877, Par., 1879, 6 s., IV., 175; Du D. chez l'enfant. Thèse Paris, 1878.—Roser, W.: Diabetes und Sepsis. D. med. Wochenschr., Berl., 1880, VI., 1, 13.—Seegen, J.: *a.* Ueber den Einfluss des Karlsbader Wassers auf D. m. Wien. med. Wochschr., 1875, Nr. 13; *b.* Zur Frage über das Vorhandensein von Zucker im gesunden menschlichen Harn. Wien. med. Wochenschr., 1878, Nr. 12 u. 13.—Seegen u. Kratschmer: Pflüger's Archiv f. d. ges. Physiol., XIV., S. 593.—Senator: Ueber die Ausscheidung des Kreatinins bei D. m. und ins. Virch. Archiv., LXVIII., p. 422.—Schmitz, R.: Hochgrad. Insuff. der Herzthätigkeit u. s. w. Berl. klin. Wochenschr., 1876, Nr. 5.—Solomon, G.: Untersuchungen betreffend d. Vorkommen von Glycogen u. s. w. D. med. Wochens., 1877, Nr. 8.—Sanders and Hamilton, D. J.: Lipæmia and Fat Embolism in D. Coma. Ed. Med. Journ., 1879-80, XXV., 47-52.—Stannard, I. H.: Fatal Case of D. M.; Lesion of Liver and Gall Bladder. Pacific M. and S. J., San Fran., 1879, XXIII.—Starr, Louis: Lipæmia and Fat Embolism in D. M. N. Y. Med. Record, 1880, XVII., No. 18.—Teschemacher: Ueber Aetiologie des D. m. Diät u. Curerfolge. Ahrweiler, 1877.—Tessier, L. J.: Du D. phosphatique. Paris, 1877.—Taylor and Goodhart: Guy's Hosp. Rep., 1877, XXII., p. 415.—Ulrich, F.: Sur le diabète. Paris, 1879.—Veit, O.: *a.* Zur Diagnose des D. m., insbesond. der Anfangsstadien. Berl. klin. Wochenschr., 1876, No. 41, etc.; *b.* Coma Diabeticum. Berl. klin. Wochenschr., 1879, XVI., 31-33.—Vulpian, A.: Diabète sucré. Chir. méd. de l'Hôp. de la Charité. Paris, 1879, 500-504.—Verneuil et Lucas Championnière: Rapport sur un travail, intitilé; Note pour servir à l'histoire des lésions traumatiques chez les diabétiques, par Gabriel Maunoury. Bull. et Mém. Soc. de Chir. de Paris, 1879, n. s., V., 755. —WINCKEL, F.: Erkrankungen der äusseren Genitalien. D. Ztschr. f. p. med., 1876, Nos. 1-8.—ZIMMER, K.: *a.* Levulose im Harn eines Diabetekers. D. med. Wochenschr., 1876, No. 28; *b.* Erythema chron. bei D. Prag. med. Wochenschr., 1876, Nr. 17.—ZÜLZER: Virchow's Archiv, LXVI., pp. 223 and 282.

Heretofore the credit of first having discovered sugar in diabetic urine has been given to *Thomas Willis.* To him it belongs, although the London *Lancet* has called attention to the fact that *Paracelsus* in his "De Morbis ex Tartaro ariundis," more than a hundred years previous, noticed "a peculiar solid residue" in this urine.

ÆTIOLOGY.*

Diabetes occurs with very marked frequency in southern Italy and Malta according to *Cantani.* It is of very rare occurrence in Western Thuringia and in the Thuringian Forest. The following table compiled by *Pfeiffer* is of value in showing its relative frequency:

1 Death from D. m. is proportional to:			1 patient with D. m. to patients in general.	
	DEATHS IN GENERAL.	INHABITANTS.		
Weimar	1:200	1:11250	Jena Clinique	1:322
Frankfort a. M.	622	27300	Würzburg Clinique	637
Chemnitz	977	32000	Apolda Clinique	1750
Hamburg	1172	38166	Barmen (Hospital)	15000
Brooklyn	1947	86555	Schmalkalden	17000
New York	1519	50585		
England	1909	86000		
Seven Rhenish cities	2110	45743		
Erfurt	4000	140000		
Kingdom of Bavaria	4290	128000		

In regard to its origin from hereditary influences, it may be said that additional cases in children have been reported by *West, Pfeiffer, Risel, Cantani,* and *Redon. Dumontpailler* treated a child whose ancestry suffered from gout, and he claims that this disease exerts an hereditary and predisposing influence upon d. m. This is still further affirmed by *Lancereaux* (1879), who relates three or four cases. An increase of the statistics to one hundred and thirty-two cases of diabetes in children under fifteen years of age has left the preponderance, according to *Senator,* still in favor of the female sex. Of one hundred and thirty-two cases, sixty-one were in boys and seventy-one in girls.

It has been noted that d. m. occurs more frequently in the well-to-do than in the poorer classes, and of two hundred and eighteen cases *Can-*

---

* We are so constantly indebted in the articles upon diabetes to the very able writings of *Senator* in the second edition of *v. Ziemssen's* Cyclopædia that we wish in advance to make acknowledgment of a very considerable reproduction from this author. For the sake of convenience, we have omitted quotations marks in many places.

tani reports one hundred and nine gentlemen of leisure, clergymen and lawyers. "A sedentary mode of life, with little muscular exertion and with a hearty diet, bears a particularly important relation."

As to diet, it is believed by *Cantani* that the excessive farinaceous and saccharine diet of Italy causes the prevalence of the disease. He has found it very common among negroes on sugar plantations.

Of the causes, in addition to traumatisms, to nervous disorders, to psychical influences and others that have always been accepted, *Senator* considers as now established that disturbances of digestion play an important part in originating the disease in many cases. And that, after this disturbance exists, any unusual introduction of sugar or sugar producing substances into the system will hasten the outbreak of the disease or increase its symptoms so as to lead to its recognition.

The tendency at the present time is to accept the existence of "two stages" rather than "two forms" of diabetes m. This view has been well maintained by *Traube*, *Dickinson*, *Bence-Jones*, and *Senator*. The two stages, an "early" and a "late" stage, are recognizable by a diminution in the excretion or disappearance of sugar from the urine, when the patient is placed upon a strictly nitrogenous diet. When this takes place, the patient is considered in the "early" stage; when, however, in spite of such a diet the excretion of sugar continues, the "late" stage is said to be present. It is very certain that diabetes frequently exists for some time before it is recognized, and if we remember this the temptation disappears to consider cases made apparent in the "late" stage, as of a "severe form." Of course, exceptionally, as in children, we meet with what might be called "acute diabetes," in which the disease is at once severe or manifests the "late" stage only and proves rapidly fatal.

PATHOLOGICAL ANATOMY.

We must still admit that as yet no constant lesion has been found. Changes in the liver have been more frequently met with than in any other of the viscera, but these are too varied to permit of a definite conclusion being drawn. In a case reported by *Frerichs* recently, he found islets of diseased tissue in an otherwise healthy gland, in which the interlobular veins and capillaries were considerably dilated and the liver-cells atrophied.

Cantani has noted a peculiar change in the kidneys. The epithelium of the straight tubules of the cortex was the seat of a watery infiltration and swelling, converting the tubules into transparent, hyaline bladders. The vessels of the glomeruli and interstitial capillaries were also dilated.

Lancereaux very recently emphasizes the significance of the lesion in the pancreas, claiming that he has met it in his severe cases, in which autopsy was held.

Cantani has stated that the sugar found in the blood of diabetics is distinguished from urine sugar (glucose) by its inability to rotate polarized light, and he has called it "paraglucose." This statement has been shown to be incorrect by recent repeated observations of *Külz* and *v. Mehring.*

In the nervous system, the lesions claimed by *Dickinson,* of dilatation of the arteries and perivascular spaces, have not been confirmed by *Taylor* and *Goodhart,* and *Frerichs* failed to find them in many diabetics.

De Giovanni found in one case fatty degeneration and softening of the vermiform process and sclerosis of the right lobe of the cerebellum. *Frerichs* reports a case with atrophy, and diminished number of the ganglion cells in the anterior horns and *Clark's* columns. In both cases dilatation of the central canal of the spinal cord existed, and in the latter the canal was choked with lymphoid cells. *De Giovanni, Ponicklo,* and *Cantani* saw changes in the sympathetic ganglia. All three noticed atrophy of the nervous elements. In addition to this, *De Giovanni* reports pigmentation and thickening of the capsule, also changes in both vagus nerves, and *Ponicklo,* sclerosis of the upper and lower cervical ganglia. *Cantani* believes the atrophy *in the solar plexus* which he saw in his cases to be result of the marasmus.

SYMPTOMS.

URINE.—*Lecorché* states, concerning the relative quantity of urine passed during the night and the day, that in the early period of the disease, the evacuation of urine is generally copious at night, especially with an exclusively nitrogenous diet, but does not exceed that passed by day. In farther advanced disease, with a starchy diet, the amount discharged by day is greater than that by night, while the reverse holds on an animal diet. *Gorup-Besanez* called attention to a copper-reducing and left-rotating substance—probably levulose—in the urine of diabetics. Since then *Zimmer, Czapek,* and *Redon* have investigated this substance. Levulose responds equally with glucose to the copper tests; its rotary power examined by the polariscope is much influenced by temperature. *Czapek* obtained 39.4 pts. levulose and 60.6 parts glucose in his case, *Zimmer* 9.8 per cent glucose and 2.2 per cent levulose. Considerable difficulty exists in coming to a conclusion whether it be really levulose. Chemically, it is well known that levulose cannot be derived from the starches, but only from saccharose itself (also from inulin). Hence in the majority of cases it could not be derived from the food. Still further, recent investigations made by *Haas,* and confirmed by *Johannowsky,* have demonstrated the presence of a left-rotating and copper-reducing substance in normal urine, in small quantity.

Inosite which has repeatedly been found, was met in five of thirty-eight diabetics by *Gallois,* and he had inosite and glucose appear alternately in a rabbit, upon irritation of the medulla after the manner of *Bernard. Reichhard* reports a case in which dextrine alternated with glucose. Creatinin is increased in diabetic urine, according to *Senator.*

Zülzer and *Frerichs* have determined the relative proportion of nitrogen and phosphoric acid in diabetic and healthy urine to be the same. *Czapek* in two cases found the quantity of phosphoric acid relatively smaller in the night urine of the diabetic, but also observed it to increase with the improvement of the patient.

Tessier has called attention to another relation of the phosphoric acid discharge to sugar excretion. He has met with cases in which the glycosuria alternates with phosphaturia or increased excretion of phosphoric acid, the symptoms of diabetes continuing as before, giving rise to an affection which he calls diabetes phosphaticus. *Senator*, judging from a case of his own, considers the subject important and worthy of further investigation.

Bussard has confirmed *Tommasi's* view that the spermatozoa are killed by the blood sugar. He observed, in the case of a physician suffering from transitory diabetes, that the spermatozoa which during the disease were absent or few and motionless, were found in abundance and alive soon after the cure of the diabetes.

Among the fungi *Hallier* and *Kuessner* have found chains of the leptothrix in the fresh and stale urine, and in the bladder of diabetics.

ACETONÆMIA.—DIABETIC COMA.—Of late years a peculiar mode of death has frequently been met with, to which these names have been given. It is well and briefly described by *J. Cyr* as follows: Three principal stages are observed with considerable constancy; first, excitement, second, dyspnœa, and third, coma. "The period of excitement is manifested by some incoherence, vivacity, and rapidity of speech with some indistinctness, vague malaise and disquietude, going on even to anguish. To this succeeds difficulty of breathing, occurring suddenly and sometimes voluntarily; deep expirations made with effort, the thoracic muscles acting vigorously, the lungs dilating, but the oppression persisting nevertheless. This, the most characteristic stage, is followed by exhaustion and coma, and death sometimes occurs in twenty-four hours, sometimes in less time."

In cases terminating in this manner, it has been frequently observed that acetone was present in the breath of the patient, and occasionally in the urine. Still further it has been noted that with the approach of the above symptoms and during their continuance the excretion of sugar decreased very markedly. It has therefore been presumed that, in the first instance, acetone was being stored up in the blood, in the latter that glucose was accumulating, and the names acetonæmia and hyperglycæmia have been proposed. Except a dark thick condition of the blood, no constant and striking condition of the blood has been found at autopsies, to which the symptoms described and the death could be referred. In one case, *B. Foster* found on microscopic examination of the blood numerous fat-like granules. These did not, however, dissolve in ether, and he ascribed them to the action of acetone, since at other times autopsical blood treated with acetone produced similar granules. *Sanders* and *Hamilton* have recently failed in obtaining this latter result, and believe the granules to be oil. As to the source of acetone, the recent experiments of *Foster* have confirmed the idea that it is a product of fermentation of grape sugar. And it is generally held that indigestion and stomach fermentation will readily give rise to its formation. Acetone, as is known, is met with in the expired air, in the blood, in the urine, and after death recognized in the stomach and intestines. That the odor of acetone is not perceptible in the breath of all these cases of diabetic coma is explained by *Foster* by the fact that acetone has a high boiling point (58° C.). The odor is, therefore, more readily perceptible in feverish diabetics. But we doubt the acceptability of this explanation. Very recent investigations by *Quincke* cause him to doubt whether the substance which *Geuther* first demonstrated as present in the urine, and which he called æthyl-diacetic acid, but which *Quincke* calls by the name acetic æther, is always present in the urine. He thinks the test (that by Fe_2Cl_6) may give a similar response for a substance "æthylaceto-acetate," and thinks that this latter may sometimes be present. Still further, in corroboration of his doubts, in experiments upon animals in which he injected acetic æther, he failed, except in a few instances, to find it in the urine. He believes, therefore, that substances other than acetone are also formed, and in offering an explanation for the symptoms of diabetic coma, he concludes "it appears to me that a strong analogy exists between these nervous phenomena in d., and the similarly varying picture which uræmia presents; here as there it is not *one* but *several* substances which are formed and heaped up in changing amount by the abnormal tissue changes and give rise to symptoms of poisoning, which, though considered as belonging to one type, vary more in individual cases than the symptoms, for instance, of poisoning by an alkaloid."

It is quite likely then that other substances besides acetone of the ethyl acetic series will be demonstrated to be formed in the blood, as *Quincke's* investigations already tend to show. To accept the term acetonæmia and to admit that the symptoms are due to the presence of acetone, which *Senator*, *Lecorché*, and *Cyr* have done, is as yet hardly justifiable (see also *Lancet*, 1878, p. 689). Very recently (July, 1879), Prof *Sanders* and Mr. *Hamilton* suggested a new theory, that the symptoms of diabetic coma (so-called acetonæmia) were the result of carbonic acid poisoning caused by fat embolism in the lungs. This view was based upon the examination of several cases in which they found emboli in the lungs, also in the kidneys and other organs; the close resemblance in histological appearances of the lungs and of the symptoms caused by fat embolism from fracture; the milk-like appearance of the blood in their cases, which they proved to be due to the presence of oil. The source of the embolism was accounted for by the existence of oil in the blood. Since this publication by *Sanders* and *Hamilton*, *Starr* has published an additional case of death from diabetic coma, in which the presence of fat in the blood, of marked fatty embolism in the lungs, and of slight fatty embolism of the liver and kidneys was admirably demonstrated. Unfortunately in this case the patient also suffered from pneumonia. It is, therefore, difficult to say how far the fatty embolism assisted in producing the symptoms and death. The data for the theory of *Sanders* and *Hamilton* are as yet too few to make its acceptance justifiable. They have, however, called our attention to the subject, and future investigations may add to the number of cases of death from fatty embolism.

PHYSIOLOGY.

v. Mehring, by a series of well conducted experiments, has demonstrated that the statement of *Bernard* "that the blood of the portal vein is entirely free from sugar" is to be corrected. He has found no difference in the quantity of sugar contained in the blood of the portal vein, hepatic vein, and other vessels. Recent investigations upon the lower animals by *v. Mehring, Wolffberg* and *Finn*, and observations upon diabetics by *Külz, Frerichs* and *Adamkiewicz*, corroborate *Bernard's* conclusion that albumen belongs to the glycogen producers. According to *Seegen* and *Kratschmer*, no special ferment is needed for the conversion of glycogen into sugar, since even small quantities of any albuminous substance can effect it.

v. Mehring has also found nearly equal quantities of sugar in chyle, whether the animal had been fasting or fed upon meat or with sugar and starch. The old idea that the sugar of the chyle is derived by absorption from the intestine is, therefore, not tenable. The lacteals therefore take no important share in the absorption of sugar.

The question whether sugar is present in normal urine is still a subject of divided opinion. *Külz* failed to obtain it in demonstrable quantity, even in 100 litres. *Malygin* states that he has detected .0032–.014 per cent of sugar, and *Pavy* (Guy's Hosp. Rep., 1876) has found .0059 per cent.

In some very recent experiments of *Pavy* conducted, he claims, with unusual care, he failed to find glucose in the liver and comes to the following conclusion: "the liver is essentially a sugar assimilating, instead of a sugar forming organ, and that when its assimilative action is properly exerted, so little sugar is allowed to pass into the general circulation that the quantity existing in arterial blood is insufficient to render the urine more appreciably saccharine than is observed in the healthy state; but that when its assimilative action is not properly exerted, sugar is allowed to pass, and in proportion as it does so, the urine acquires a more or less marked character." It appears to us that further investigations must be made, before we are ready to set aside such convincing experiments as those of *Dalton* in proof of the fact that glucose is formed and found in normal liver tissue.

The influence of the nervous system upon the formation of glycogen has been recently well demonstrated in experiments upon rabbits by section of the spinal cord by *J. Mayer*, and by irritation of the vagus (Nv. depressor.) by *Filehne, Külz* and *Eckhard*. The latter, by irritating the vagus, constantly produced glycosuria. *Mayer* found (1) whether the cord be divided between the fifth and sixth cervical vertebræ or between the last cervical and first dorsal, or between the second and third dorsal, grape sugar introduced directly into the veins of the body is in part retained in the body as such or as glycogen; (2) Division between the fifth and sixth cervical vertebræ has an inhibitory action upon the formation of glycogen (in the liver) from the sugar introduced into the circulation, without increasing the excretion of sugar through the urine; (3) Division between the last cervical and first dorsal increased the formation of glycogen (in the liver) from the sugar introduced into the circulation, without the sugar of the blood being diminished, and that (4) Division between the second and third dorsal vertebræ diminished the formation of glycogen (in the liver) out of the sugar introduced into the circulation and produced a considerable consumption of the same in the body.

Vulpian presented the results of the experiments of *Dastre*, which prove that asphyxia will produce glycosuria, instead of causing the sugar of the body to disappear as *Cl. Bernard* maintained.

Cases of diabetes from absorption of milk sugar are met with, but they are not true cases of diabetes, rather cases of "lactosuria." Lactose differs both in its rotating power and copper-reducing action from glucose.

Cantani in his recent work defines D. m. "as an anomaly of metamorphosis, in which, without the occurrence of a quantitative or qualitative abnormal formation of sugar, the sugar introduced or produced in the body from albuminates is not used," and is therefore discharged in the urine. *Senator* believes, however, that "the incapacity to utilize the sugar is not sufficient by itself to explain diabetes. This view is opposed by the behavior of many light cases of the disease towards different kinds of food. Many patients of this kind can consume and digest very large quantities of albuminates without excreting sugar, while the smallest amounts of sugar or starch in the food causes its excretion. In both cases the diabetic receives or forms carbohydrates, for we now know that glycogen is formed under the influence of albuminous food, and that when much albumen is ingested the amount of glycogen (and sugar) formed in this way is certainly greater than on a scanty diet of sugar and starch. If now sugar is excreted in the former case, and not in the latter case, the cause cannot be or not alone a diminished capacity on the part of the organs, especially the muscles, to convert the carbohydrates brought to them by the blood, but only in abnormities in the pathway from the alimentary canal to the general circulation. The changes which may here be claimed have already been stated, and it is now only necessary to mention that this difference may be explained, if we suppose that in the lighter cases ("first stage," "light form") changes occur only in the alimentary canal, through which the ingested or starch-produced sugar has been to an abnormally large degree unchanged, that is, has passed into the circulation without having been transformed into glycogen; on the other hand, in the severer cases ("second stage," "severe form") a more rapid or more abundant conversion into sugar of the glycogen produced from albuminates has taken place. Besides this, the capacity to utilize the sugar which has passed into the blood may be more or less diminished in diabetics, and indeed, considering the behavior of the milder cases with abundant albuminous diet, the severe cases alone or in particular must have lost this capacity."

We may finally mention that disturbances of the alimentary canal seem to have an increasing importance in the pathogeny of diabetes, (1) in so far as they directly change the digestion of carbohydrates, and (2) since they may excite the excretion of sugar by reflex action.

DIAGNOSIS.

As yet the elements in the diagnosis of diabetes remain the same as in the past. The tests for sugar have not been improved upon by any of the recently proposed modifications. The copper reduction by the use of Fehling's solution is still the most reliable test we have. A few practical suggestions in the application of this latter test might here be made. The use of a porcelain capsule instead of a test tube enables a quantitative test to be made with greater accuracy, for we can more readily recognize if any unchanged copper solution be left, the slightest blue tint showing itself readily by contrast with the white porcelain. A still greater degree of accuracy will be attained if, after we are no longer able to distinguish any blue color, we remove a small portion of the clear solution in a small test tube, add to it a drop or two of glacial acetic acid and then a few drops of a very dilute, nearly colorless solution of ferrocyanide of potassium. If any copper be still in solution and unreduced, we will obtain a characteristic red precipitate of ferrocyanide of copper.

There is but one substance, which is infrequently found in human urine (*Baumann*), which can give a similar reaction to the copper tests for sugar, this is pyrocatechin. It is recognized by the very dark color which the urine assumes upon the addition of an alkali (*Bödeker, Ebstein, Müller, Fürbringer*). When present, it is to be eliminated by the addition of plumbic acetate to the urine, and the filtrate is to be used for titrating.

The polariscope does not always furnish the data for a quantitative determination of sugar. For if an inactive or left rotating substance be present it becomes unavailable, or can only serve to direct attention to other rotating substances, when its results differ considerably from those obtained by titration.

It is interesting and worthy of remembrance that various methods of deception on the part of patients are resorted to. *Abeles* and *Hoffmann* relate a case in which sugar solution was injected into the bladder.

DURATION.—*Redon* found 2½ years to be the longest duration in 32 diabetic children. *Külz* furnishes the following table of duration in 46 children:

Less than ¼ year, . .	16
¼ " . .	8
½ " . .	6
1 " . .	5
2 years, . .	6
3 " . .	4
4 " . .	1
	46

Thus more than half run their course in three months and less.

COMPLICATIONS.—*Senator* states that general tuberculosis has repeatedly been met with in diabetics.

Schmitz has found objective signs of weakness of the heart in 80 of 109 diabetics.

Eye Affections.—In addition to cataract, though not so often, we meet with retinal disease, affections of the optic nerve and of the ocular muscles. The retinitis of Bright's disease—hemorrhagic retinitis with white foci of degeneration, and retinitis complicated with optic-nerve atrophy (*Galezowski*) are met with. Simple amblyopia, optic-nerve atrophy and hemianopsia form the optic-nerve lesions; and paralyses of the ocular muscles, which, according to *Förster*, may be produced by hemorrhages into the nerve sheaths, are frequently observed. Frequent opacity of the vitreous has likewise been noted. *v. Graefe* referred to the occurrence of suppurative keratitis in diabetes, though he considered it the result of the general malnutrition (compare *Leber*, Ueber die Krankheiten des Auges bei D. M. in v. Græfe's *Archiv f. Ophthalm.*, XXI., Abt. 3, and in Krankh. der Netzhaut und des Sehnerven in Graefe's und Saemisch's Handb. d. Augenheilk., V., pp. 395 and 893, and *H. Cohn*, Amblyopie und Augenmuskellähmungen bei D., in *Arch. f. Aug. und Ohrenheilk.*, 1878, VII., Abt. 1. p. 33).

Cutaneous Affections.—*Winckel* describes three kinds of cutaneous lesion of the female sexual organs in glycosuria: (1) Simple mycosis in the form of white specks on the vulva, of the size of the head of a pin; these specks containing a fungus leptomitus, which is identical with mycosis of pregnant women, and is not produced by the saccharine urine. (2) Furunculosis labiorum. (3) Phlegmonous vulvitis of the severest form, which can attain a great extent, and at an early period, sometimes before demonstrable evidence of sugar is found in the urine. Fungi in this form are scanty or not found at all. *Hardy* has sought to explain the occurrence of erythema met with in the suprapubic fold by the contact of the parts with the saccharine urine, but he has seen the affection occupy regions of the body which the urine had not reached. This latter experience is corroborative of *Winckel's* view and strongly opposes the belief of *Haussman*—that the fungi are produced by the saccharine urine, and are the cause of the inflammations. In males, *Hardy* has frequently met with balanoposthitis, accompanied by intolerable itching.

Gangrene following operations is a common accident, and idiopathic gangrene likewise occurs. The value to be attached to these lesions in their relation to diabetes is readily appreciated when we recall the statements of *Hardy* and *Roser*. The former says: "It has quite frequently happened to me to diagnosticate diabetes in women by the existence of erythema in the suprapubic fold." The latter gives as his experience that, when an individual, apparently in good health, suffers from a progressing gangrenous or ulcerating lesion, say on the extremities, when there seems to be no infecting cause, and when all irrigations with antiseptics prove useless, it is "high time" to examine for diabetes.

Pregnancy.—A few cases are reported which tend to establish a relation between pregnancy and diabetes, but evidence is insufficient. It is noted, however, that diabetics are inclined to sterility.

TREATMENT.

The importance of proper dieting being always appreciated by every investigator of this subject, as successive theories in regard to it prove a failure, new ones are constantly suggested to accord with the change in views. Thus, *During* recommends a plan of treatment in which the patient shall receive three or four meals daily, at regular intervals, and is to consume in all, daily, from 80–150 grm. of rice or barley, 200–500 grm. of meat, in addition to some milk and wheat-bread, with fruit and a glass or two of red Rhine or claret wine. The patient is also to seek to harden himself by gymnastic exercises, shampooing, manipulating, etc. *Cantani's* plan, based upon the idea of giving rest to the "diseased organ" by replacing the sugar and carbohydrates that are not utilized by albuminates and fat, and thus gradually to eliminate the sugar from the blood, recommends an absolute meat and fat diet. He forbids the use of spices, of sugar or starch in its strictly chemical signification, which excludes liver, milk, butter, cheese, plants with their juices and infusions, even vinegar in severe cases. An occasional fast-day is also added in intractable cases.

Bouchardat has recommended a bread prepared by *D. J. Cormier* ("pain de gluten et légumine) in which the wheat flour is replaced by bean flour, because the flour of beans contains relatively more nitrogen (vegetable albumen) than ordinary flour. For soups, a preparation—"glutervine"—is also made from gluten and lentil flour. A serious objection to these preparations, as also to the almond-biscuit and similar manufactures, is that they are difficult of digestion and that diabetics, who are almost always dyspeptics, are made to suffer considerably by their use. As a substitute for bread, the biscuit made of bran is still the one most agreeable and least disturbing to the majority of patients.

Frequent attempt is made to arrive at a correct conclusion as to the value of various mineral waters and places of resort in diabetes.

The much extolled Carlsbad is still recommended by *Senator*, though experiments with its constituents upon diabetics failed to produce any amelioration. The opinion of *Senator*, that its benefit depends only upon the fact of the water being warm and that it is drunk in considerable quantities, seems strongly corroborated by the experience of *Glax*. He improved two cases very decidedly by having them drink from 1000–1400 ccm. of warm distilled water daily.

With a view to replace sugar (as in coffee), *Martin* (Bull. de Thérap., Sept., 1877), recommends an infusion of licorice.

Cantani and *Pawlinoff* recommend the use of lactic acid. *Lauder Brunton* suggests that it be combined with bicarbonate of soda (five to six grammes), in which combination even large amounts of the acid are well borne. *Cantani* believes that it aids digestion of albuminates; *Pawlinoff* that it protects the albuminates of the body against oxidation by furnishing in itself a substance so much more readily oxidizable.

Bouchardat has seen the sugar entirely disappear in diabetics who excreted but little sugar, when glycerin was daily administered. He strongly recommends its use in thin (and constipated) diabetics.

The use of preparations of ammonia is destined to take an important rank. As well by theory and practical experience as by experimentation, *Adamkiewicz* has proven that they are assimilated to a great degree, and that, under their use, the sugar diminishes and even disappears. The daily administration of a quantity, gradually increased to twenty grm., of the carbonates of ammonia, or of the compounds with organic acids (ammonium aceticum) is recommended.

Especial care of the teeth and mouth is to be urged, and frequent rinsing with a solution of borax and glycerin, or with alkaline and antiseptic substances.

Kussmaul, Hilton, Flagge, and *Taylor* have made use of transfusion in cases of diabetic coma, without success. *Foster* recommends opium and carbolic acid. *Roser* has proposed that, previous to undertaking any operation upon a diabetic with a view to avoiding subsequent gangrene, the patient be first put upon a dietetic regimen and the excretion of sugar reduced to a minimum. *L. Fischer* makes the additional suggestion of administering carbolic acid for some time previous to the operation.

DIABETES INSIPIDUS.

BIBLIOGRAPHY.

Blau, L.: Ueber Diab. mel. u. D. insip. (review). Schmidt's Jahrb., CLXVIII., CLXXIII., CLXXV., CLXIX.—Biermer: *a.* See Korach; *b.* Nach Lewin, Charité-Annalen, II., 1877, p. 653–656.—Charcot: Diabète non sucré, suite d'un coup sur la tête. Gaz. hebd., 1860, Feb. 3d.—da Costa: Diab. Insip. and its Treatment by Ergot. N. Y. Hosp. Gaz., 1878, Feb. 15th.—Eade, P.: Cases of Diab. Insip. Beale's Archiv of Med., 1860, II., p. 5.—Fischer, H.: Ueber die Commotio Cerebri. Volkmann Sam. klin. Vorträge, No. 27.—Frerichs: Prager Vierteljahrschr., 1875, CXXVII., p. 12.—Korach, S.: Beiträge zur Pathol. u. Ther. des

D. insip. Diss., Breslau, 1876.—Kuelz, E.: Gerhardt's Handb. der Kinderkrankheiten, III., 1878.—Kennedy, H.: Cases of Diab. Insip. and the Treatment, etc. The Practitioner, 1878, XX., 94.—Kahler: Prager med. Wochenschr., 1877, Nos. 33 and 34.—Laycock: Beneficial Use of Jaborandi. The Lancet, 1875, II., No. VII.—Lecorché: Diab. mel. et insip. Compte rend. Acad. d. sc., Paris, 1880, XC., 426.—Müller, W.: Om Diab. insip. Norsk. Mag. f. Laegeridensk., Christiania, 1879, IX.—Murrell: On a Case of Diab. Insip. Treated by Belladonna and Ergot. Brit. Med. Journ., 1876, Jan. 1st.—Mielecki, St. v.: Ueber Diab. insip. Diss., Berlin, 1875.—Ollivier, A.: De la Polyurie et des var. de la quant. de l'urée à la suite de l'hémorrhagie cérébrale. Archiv de phys. norm. et path., 1876, p. 85.—Ralfes: The Lancet, 1876, I., No. IX.—Ringer, Sidney: A Case of D. Insip. Treated by Jaborandi and Ergot. Brit. Med. Journ., 1875, p. 775.—Whittle: On Renal Diphtheria. Dubl. Quart. Journ., 1867, Nov.

ÆTIOLOGY.

According to *Ollivier*, *every* cerebral hemorrhage, irrespective of the seat of the lesion, causes polyuria for a time. Even if the portions of the brain which control the secretion of urine are not directly involved, they are affected by irritation transmitted from a distance (shock).

It is interesting as bearing upon *Bernard's* experiments of producing polyuria by irritating the vagi, that *Ralfes* states he has observed polyuria in cases of aneurism of the arch of the aorta.

SYMPTOMS.

Unlike diabetes mellitus, virility is rarely affected in this disease. *Trousseau* has related an exceptional case of impotency.

When children suffer from the disease their physical growth is apt to be seriously interfered with. This has been the observation of *Senator*, who relates a case given by *Strauss*, which occurred in a young man nineteen years old. He had suffered for eleven years, and in appearance looked like a lad ten years of age, having a high pitched voice and none of the signs of puberty.

A diminution in the severity of the disease is frequently observed shortly before death.

PATHOLOGICAL ANATOMY.

In addition to the multiple lesions already presented, *Frerichs* has met with cysticerci of the brain: the floor of the fourth ventricle was not involved. *Biermer* had one case in which multiple capillary hemorrhages of the brain with general hyperæmia existed. *Lendet* relates a case in which, in addition to changes in the meninges of the brain, he found softening of the left crus cerebri and yellow discoloration of the floor of the fourth ventricle.

DIAGNOSIS.

To make a differential diagnosis between true primary polydipsia and diabetes insipidus with secondary increase of thirst, *Kuelz* suggests to note the effect upon the disease when all liquids are withheld. In primary polydipsia the secretion of urine should be influenced just as in of the health when liquids are allowed or withheld; whereas in diabetes insipidus we have a constant abnormally large flow of water from the kidneys.

TREATMENT.

Ergot and ergotin continue to be recommended, and have proved of service in the hands of *Tillard, Foster, da Costa, Sidney Ringer,* and *Rendin.* Jaborandi too has been successfully employed by *Laycock, Kahler, Fronmüller,* and *Senator.* Benefit was obtained by the use of pilocarpine in one case by *Kahler,* and in one case *Senator* failed to benefit his patient by its use. *Biermer* reports a case of permanent cure resulting from the prolonged use of carbolic acid. Very recently *Kennedy* has recommended dilute nitric acid (beginning with one drachm per day and increasing to five). He reports five cases of cure, and looks forward to equally good results in the future.

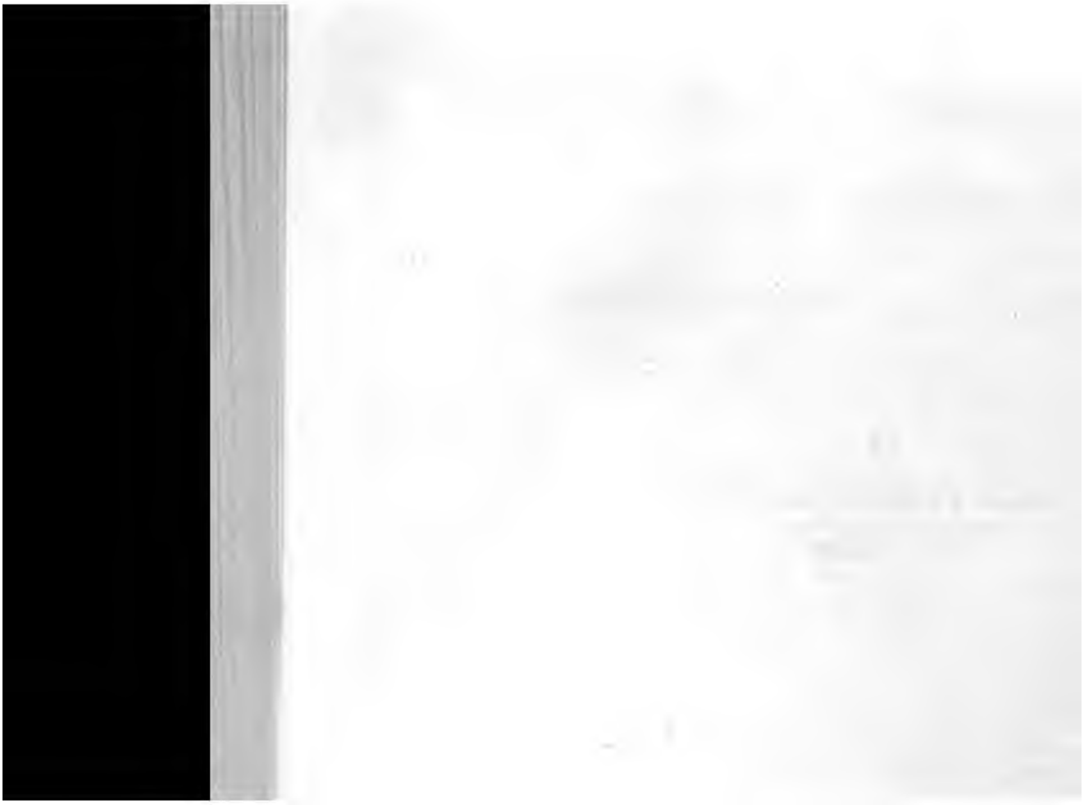
HÆMOPHILIA.—SCURVY.

MORBUS MACULOSUS WERLHOFII.

BY

H. M. BANNISTER, M.D.,

OF CHICAGO.



HÆMOPHILIA. HEMORRHAGIC DIATHESIS.

(*Hæmorraphilia. Bleeder Disease. Hemorrhagic Idiosyncrasy.*)

BIBLIOGRAPHY.

Simon, F.: Recherches sur l'Hémophilie. Paris, 1876.—MacCormac, W.: On Some Cases of Bleeders. St. Thomas Hosp. Rep., N. S., VI., 1875, p. 111.—Lossen, H.: Die Bluterfamilie Mampel bei Heidelberg. Deutsche Zeitschr. f. Chirurg., Bd. VII., 1876, p. 358.—Jenner, Sir W.: A Case of Hæmophilia. Lancet, 1876, p. 716.—Kehrer, F. A.: Die Hæmophilia des weiblichen Geschlechts. Archiv f. Gynäkol., Bd. IX., 2, 1876, p. 201.—Legg, W.: A Case of Hæmophilia (Med. Chir. Soc.). Lancet, 1876, p. 856.—Langelaan, G.: Nederl. Weekbl., 1875, p. 113.—Epstein: Oester. Jahrb. f. Pädiatrie, VII. B., 1876, p. 119.—Grandidier, L.: Die Hämophilie oder die Bluterkrankheit, 2te Aufl., Leipzig, 1876. Also, Die neueren Leistungen im Gebiete der Hämophilie. Schmidt's Jahrb., CLXXIII., 1877, pp. 185, 304.—Mackenzie, S.: Hæmophilia with Purpura and Retinal Hemorrhage. Med. Times and Gaz., March 10th, 1877.—Ritter: Ueber das Verhältniss der temporären Hämophilie der Neugeborenen zu Blutungen im spätern Alter und zu der Bluterkrankheit. Prag. med. Wchnschr., 1877, 21, 22.—Foerster: In C. Gerhardt's Handbuch d. Kinderkrankh. Tübingen, 1878, Bd. III., 1, p. 235.—Bastard, H.: Hémophilie avec Purpura hémorrhagica. Gaz. des Hôpitaux, No. 130, 1878, p. 1034.—Boerner, E.: Hämophilie oder die Bluterkrankheit in ihrer Bedeutung für die Gynäkologie. Wiener med. Wchnschr., XXVIII., 1878, Nos. 33, 35, 37, 38.—Eppinger: Hæmophilia neonatorum. Wien. med. Presse, 1878, Nos. 25, 27.—Kidd, P.: Contribution to the Pathology of Hæmophilia. Brit. Med. Jour., May 25th, 1878.—Potain, Prof.: De l'Hémophilie et de ses Relations avec le Rhumatisme. La France méd., 1879, Nos. 1, 2. —Harkin, A.: Chlorate of Potass in the Hemorrhagic Diathesis. Brit. Med. Jour., Aug. 23d, 1879.—Mosler, Fr.: Ueber hämorrhagische Diathese u. dadurch contraindicirte operative Eingriffe bei Leukæmia und ihr verwandten Processen. Zeitschr. f. klin. Med., Bd. I., ii., 1879, p. 265.—Felsenreich, T.: Zwei Fälle von Omphalorrhagie. Wiener med. Presse, XX., 8, 1879.—Weiss, M.: Ueber das Wesen der sogenannten spontanen Nabelblutungen der Neugeborenen. Prag. med. Wchnsch., 1879, IV., p. 301.—Behrend: Purpura and Omphalorrhagia in Hereditary Syphilis. Paper read before Berlin. med. Gesellsch., in 1878. Abstr. by Dr. J. N. Hyde, in Arch. of Dermatology, Jan., 1879.—Hartshorne, H.: Article in new edition of Reynold's System of Medicine, Philadelphia, 1879, p. 904.

It will be seen from the above list, which includes most, if not all, the important memoirs published since the appearance of *Immerman's* article in the first edition of *v. Ziemssen's* Cyclopedia, that, while hæmophilia is a comparatively infrequent morbid condition, its literature is not as scanty as might have been expected. Several of these papers are, however, only re-statements or summaries of facts already known, others are histories of cases which, while valuable as adding to the general sum of our knowledge of the disorder, do not contain points of sufficient interest to call for especial notice.

As respects the etiology of the disease, two of the memoirs, at least, give noteworthy data or theories. The influence of sex on the development of the disease is studied by *Kehrer*, of Giessen, and *Boerner*, in Graz, both of whom take up the subject in its gynecological aspects. *Kehrer* shows how the disorder is modified in the female by sexual peculiarities, and lays especial stress on pregnancy, as a developing cause of the symptoms of the hemorrhagic diathesis in women in whom it had previously been latent or very little manifested. He reports three cases of his own observation in support of this view, which is, however, somewhat questioned by *Grandidier* in his abstract of *Kehrer's* article in *Schmidt's* Jahrbücher. The genital organs are the most frequent locality of the hemorrhages in females, and the special periods of female sexual life, such as puberty and the climacteric epoch, as well as the physiological accidents of pregnancy and menstruation, are sometimes accompanied with notable hemorrhagic symptoms.

Boerner relates a very interesting history of a bleeding family, in which the females were all markedly affected, as well as most of the male members. The mother was one of three sisters who, without any known hereditary predisposition, were similarly affected with chronic hemorrhage from the genital organs. The conclusions of his memoir are worth reproducing; they are: (1) Hæmophilia occurs more frequently in women than has been heretofore supposed. (2) In girls there is often a sort of latency of the diathesis, which only becomes manifest from the action of definite causes, apparently connected with the reproductive function. (3) The menses of hæmophilic individuals are not normal as to quantity. (4) Coitus may be the cause, in such persons, of profuse and even fatal hemorrhage. (5) During pregnancy, serious hemorrhages may be caused by the hemorrhagic diathesis, explainable by the special local conditions connected with that state. (6) There is no special tendency to premature termination of pregnancy in hæmophilic women. (7) Delivery is always accompanied with serious hemorrhage in these cases. (8) The puerperal period in them is particularly critical, on account of the severe and often lethal hemorrhages; but it is not yet certain that the measure advised by *Kehrer*, of inducing premature birth, is on this account advisable. (9) During lactation there may be serious bleeding from the genitals, as well as from the nipples at each application of the child. Fissures of the nipple are likewise slow to heal. (10) The menses, on their re-appearance after pregnancy, are generally more profuse than before. (11) The change of life may be accompanied with serious hemorrhages, which may on the one hand render the completion of this period later, or, on the other hand, cause it to terminate fatally. (12) Very slight mechanical causes, even the shock produced by locomotion, and even psychic impressions, may give rise to obstinate hemorrhages from the genital organs. (13) In simple hæmophilia, examination of these organs, even during severe hemorrhage, reveals nothing abnormal except a tender spongy condition of the mucous membrane with patches of congestion. (14) A successful treatment of these cases has not yet been learned; we are limited to ordinary hæmostatic measures.

Boerner's memoir confirms or, at least, strongly supports the view that hæmophilia may, without known cause, arise *de novo*, and affect at once all the children of a family in which the parents and their ancestors were apparently entirely sound in this respect. Both *Kehrer* and he show that the liability of the female sex to the disorder is greater than has been believed, it having been masked, as it were, by the peculiar modifications due to sex, and the resemblance of its manifestations to the not uncommon accidents of female sexual life.

The relation between the hemorrhagic tendencies in new-born infants and hæmophilia in the adult is discussed by *Ritter*, who opposes the view held by *Kehrer*, in the above-mentioned paper, that they are both essentially the same disease. He claims that the hæmophilia of infants is a special form of disease by itself, that it is confined to early life, and that it appears primarily in children otherwise healthy, but is always connected with and preceded by some other general morbid condition, especially such as are of a pronounced septic nature, and are accompanied with disturbances in the circulation. In some of the autopsies that have been made of infants dying from these hemorrhages, there have been found fungoid organisms in the blood-channels, which afford an apparent cause for the bleeding. While he does not exclude heredity, which is present in some of these cases, *Ritter* maintains that this infantile hæmophilia is secondary to other diseased conditions, the cause of which must, for the most part, be sought in faulty hygienic surroundings. In this sense the transitory hæmophilia of infants deserves, in his opinion, the designation of hæmophilia acquisita. In this connection the purpuric symptoms sometimes observed with inherited syphilis, that formed the subject of a paper by *Behrend*, may be noticed. Hemorrhage from the navel in these cases, which had already been observed by *Zeissl*, and designated "syphilis hemorrhagica neonatorum," was of less frequent occurrence than the purpura. *Behrend* held that these symptoms were due directly to the inherited syphilis, but the weight of opinion, in the discussion that followed, seemed to be that they were due rather to the resulting cachexia, and were, therefore, not directly a part of the syphilitic disease. They might occur equally well in cachectic conditions due to other causes than specific disease.

The pathology and complications are discussed by several writers. *Simon*, as reported by *Grandidier*, "rejects the theory that hæmophilia is connected with defective primary blood-crasis, gout, scrofula, cyanosis, chlorosis, and diseases of the heart or spleen, as well as that which attributes it to narrowing, fatty degeneration, atrophy, etc., of the larger arteries. He considers that theory the most acceptable which makes it dependent upon deficient capillary innervation with resulting dilatation, although this view has hitherto found few upholders." He adduces as in favor of this theory the frequency in bleeders of nervous disorders, the occasional latency of the disease, the fact that neurotics have generally been the most effective remedies, etc., etc. He considers hæmophilia not an independent morbid species, but rather the most pronounced manifestation of the rheumatic diathesis. The arguments in favor of this view are the numerous resemblances between certain phases of the two disorders and the fact that they both seem connected with the same climatic conditions. Very similar arguments are employed by *Potain* in support of the same general opinion. It may be stated here that, in the second edition of *v. Ziemssen's* Handbuch, vol. XVII., pp. 578 and 579, *Immermann* has added two or three paragraphs in which he recognizes a certain force in the neurotic theory of the pathology of hæmophilia, and relates briefly an interesting case that came under his own observation, in which the extraction of a tooth caused fatal hemorrhage in a young woman suffering from a relapse of Basedow's disease, who had had no previous hæmophilia or hæmophilic heredity.

On the other hand, *Mosler* points out the very general coincidence of the hemorrhagic diathesis with leukæmia, of which it forms at once one of the most striking and fatal features. Over fifty per cent of the cases of leukæmia that had come under his own observation were complicated with hemorrhages, and out of one hundred and fifty collected by *Gowers* this was also the case in eighty. The relationship, therefore, between disorders of the spleen and hæmophilia is very strongly suggested by these figures.

The only special contribution to the therapeutics of the disorder that has lately come under observation is that of *Harkin*, of Belfast, read at the last meeting of the British Medical Association. He recommends chlorate of potash as a remedy especially adapted for the treatment of diseases depending upon suboxidation, defective nutrition, etc., and especially serviceable for the production of healthy blood. The hemorrhagic diathesis being, as he considered, characterized by defective chemical constitution of the blood, together with abnormal thinness of the vascular coats, a vito-chemical remedy is required to restore it to its proper condition. But apart from these theoretical considerations, Dr. *Harkin* claims to have had marked success in the treatment of various phases of the hemorrhagic diathesis with this agent. He generally ordered the medicine to be taken in the dose of one ounce of the saturated solution three times daily—one ounce of the salt to the pint of water; and if iron was also indicated, he added a drachm of the muriated tincture to the mixture. Administered in this form he claimed to have had very satisfactory results from the drug in epistaxis, hæmophilia, hemorrhage from the bowels, from the kidneys, from the lungs, the stomach, in menorrhagia, in scurvy, and in purpura hemorrhagica. He claims, in fact, that its persevering administration will even eradicate the constitutional taint on which some of these disorders depend. This treatment seems theoretical rather than practical, and details of his experience, which have not been published so far as known, would be of interest.

SCURVY–SCORBUTUS.

(German: *Scharbock.*)

BIBLIOGRAPHY.

Young, A.: Ten Cases of Scurvy. Med. Times and Gaz., June 8th, 1872.—Cheadle, W. B.: Scurvy and Purpura. Brit. Med. Jour., Nov. 9th, 1872.—Mortier, J.: Diathèse Scorbutique. Presse méd. Belg., No. 35, 1872.—Da Costa, J. M.: On Scurvy. Phila. Med. and Surg. Rep., Nov. 16th, 1872.—Saunier: Six Cas de Scorbut de mer. Presse méd. Belg., No. 36, 1872.—Krugkula: Zur Ætiologie des Scorbuts. Wiener med. Wochenschr., Bd. XXIII., Nos. 26 and 27.—Kraus: Ursachen und Vorbeugungsmittel des Skorbuts. Allg. Militairärztl. Zeitung, 1873, Nos. 35–52.—Jalland, W. H.: Scurvy in a Ten Months Old Child. Med. Times and Gaz., 1873, Mar. 8th.—Champouillon: Gaz. méd. de Paris, 1873, No. 50.—Villemin: Cause et Nature de Scorbut. Bull. de l'Acad. de Méd., 1874, Nos. 32 and 33.—Leroy de Mericourt: Causes et Nature du Scorbut. Ibid., Nos. 42 and 43.—Kirchenberger, S.: Die Scorbutepidemie der Prager Garnison im Jahre 1873. Prager Vrtljrschr., III., p. 33.—Liouville, H. et Landrieux: Du Scorbut. Gaz. des Hôpitaux, 1874, Nos. 131, 132, 134, 136; and 1875, Nos. 21 and 23. —Berchon, E.: Du Scorbut; de sa Prophylaxie; de sa Contagion. Ibid., Nos. 134 and 135.—Benech: Essai Critique sur une petite Epidémie du Scorbut. Gaz. Hebdom., Nos. 46 and 48, 1874.—Ralfe: A Case of Scurvy, etc. Med. Times and Gaz., 1875, July 24th.—Stadthagen, M.: Ueber die Genese des Scorbut. Diss. Berlin, 1875.—Hattute: Note sur la Scorbut. Rec. de Mem. de méd. Milit., Jan. and Feb., 1875.—Azevedo, C.: Hist. Méd. Chirurg. de l'Escadro Brésil., etc. Arch. de Méd., Nov., 1875.—Robert: Du Scorbut dans la Dysentérie. Thèse de Paris, 1875.—Cron: Ein Fall von tödtlichem Scorbut. Berliner klin. Wochenschr., 1876, No. 48.—Scurvy in English Merchant Ships. Sanitary Records, II., p. 210.—Smith: Cases of Scurvy in the Polar Seas. Brit. Med. Jour., Dec. 16th, 1876.—De Chaumont, F.: Lime-juice as a Preventive of Scurvy. Sanitary Record, Nov., 1876.—Rae, J.: Lancet, 1876, II., p. 732.—Herr, A. J.: Transactions of the Medical Soc. of the State of Pennsylvania, XI., 1876, Dec. 16th.—Leach, H.: Brief Notes on the Outbreak of Scurvy in the late Arctic Expedition. St. Bartholomew Hosp. Rep., XIII., 1877.—Besnier: Le Scorbut dans les Prisons de Paris. Rapport à la Soc. Méd. des Hôpitaux, 1877.—Mathelin, E.: Contributions à l'Histoire du Scorbut. Journ. d'Hygiène, Aug., 1877.—Ralfe, C. H.: Researches on the Pathology of Scurvy. Lancet, June 16th, and July 21st, 1877.—Hohlbeck: Harnanalysen bei Scorbut. St. Petersb. Med. Wochenschr., 1877, No. 33.—De Beauvais: Épidémie de Scorbut à la Prison de Mazas. Gaz. des Hôpitaux, 1877, No. 88.—Revillout, V.: Specificité, Spontanéité dans le Scorbut. Ibid., No. 72.—De Lisle, F. I.: Med. Times and Gaz., Sept. 15th, 1877. —Frank, Fr.: Wiener med. Presse, XVIII., 1877, p. 1018.—Wegscheider, H.: Zwei Fälle von sporadischem Scorbut. Deutsche med. Wochenschr., III., 1877, Nos. 17 and 18.—Donnet and Fraser, T. R.: Report of the Committee appointed, etc., to inquire into the Causes of the Outbreak of Scurvy in the recent Arctic Expedition, etc. London, 1877.—Lüttich, B.: Neuere Beobachtungen über Scorbut. Schmidt's Jahrb., CLXXVI., 1877, p. 73.—Leroux, Ch.: Deux cas de Scorbut sporadique. La France Médicale, Aug. 14th, 1878.—Nicolas: Gaz. Hebdom., 2 ser., XIV., Nos. 1 and 2.—Uskow, N.: Pathologische Anatomie des Scorbuts. Centralbl. f. d. med. Wissensch., 1878, No. 28.—Cheadle, W. B.: Clinical Lecture on three Cases of Scurvy supervening on Rickets in young Children. Lancet, 1878, II., p. 685.—Magnus: Exophthalmus auf scorbutischer Grundlage. Deutsche med. Wchnschr., IV., 29, 1878.—Gallico, E.: Effusione emorragica nell addome per Diatesi Scorbutica. Gaz. Med. Ital. Prov. Venet., XXI., 1878, p. 350.—Segre, I.: Contribuzione allo Studio dello Scorbuto. Giorn. Venet. di Sci. Med., 1878, 3 ser., XXIX., p. 489.—Pinder, G.: Ueber Angina Scorbutica. Wiener Med. Wchnsch., 1878, No. 39.—Da Costa, J. M.: Diagnosis and Treatment of Scurvy. Med. and Surg. Rep., 1879, p. 75.—Mathelin, E.: De la Prophylaxie du Scorbut dans la Marine Marchande. Rev. d'Hygiène, I., 134, 247.—Scheby-Buch: Zur Unterscheidung der Contusionen von den freiwilligen Blutunterlaufungen bei den scorbutartigen Krankheiten und dem Erythema nodosum. Vrtljrschr. f. Dermatol., 1879, VI., 89.—Von Ziemssen: Scorbut. Lungenspitzencirrhose, Dilatation und Hypertrophie des rechten Ventrikels, allgemeiner Hydrops (mitgetheilt von J. Freudenberger). Aerztl. Intelligenzbl., Munich, 1879, XXVI., 223. —Da Costa, J. M.: A Clinical Lecture on Scurvy. Hosp. Gaz., N. Y., 1879, VI., 221.—Zemanek: Der Skorbut und seine militär. hygienische Bedeutung; mit spezieller Berücksichtigung der unter den k. k. Okkupations-Truppen in Bosnien und der Herzegowina aufgetretenen Skorbut-Epidemie. Militärarzt, 1879, XIII., p. 169, p. 180.—Hume, T.: Report on Scurvy and Chronic Rheumatism (Scorbutus and Myalgia), as it occurred in the 1st Infantry, H. C., at Lingangoor in 1878. Ind. Med. Gaz., Calcutta, 1879, XIV., p. 329.—Cheadle, W. B.: Rickets; Defective Diet; Development of Scurvy; Rapid Recovery on Anti-Scorbutic Diet. Brit. Med. Jour., 1879, II., 987.—Kuhn, A.: Ueber leichte Scorbut-Formen. Deutsches Arch. f. klin. Med., 1880, XXV., p. 115.—Yeld, H. P.: Report on the Health of the Troops forming the Vitakri Field Force, and upon an Outbreak of Scurvy among them. Ind. Med. Gaz., XV., 1880, 10–12.—Little: Outbreak of Scurvy at Akola, India (quoted in Brit. Med. Jour., 1879, 786—*Editorial*). —Fabre, A.: Les Relations Pathogéniques des Troubles Nerveux, Paris, 1880. Chap. XI., l'Action vaso-motrice et le Purpura dans leurs Rapports avec le Scorbut, p. 376.—Mahé, J.: Article "Scorbut" in Dict. Encycl. des Sciences médicales. Troisième série, Tome VIII., Première partie, Paris, 1880.

The above list of papers that have appeared for the most part since the publication of the first edition of *v. Ziemssen's* cyclopedia, though containing over fifty different titles, is yet somewhat incomplete, so rapidly has the literature of the subject increased within the past few years. It includes, however, most of the more important recent contributions.

The etiology and nature of scurvy being the questions of greatest interest and uncertainty, form the subject of several of the most elaborate articles and are discussed in nearly all except the purely casuistic ones. The theory that scurvy is an infectious or contagious disease has been revived by *[illegible]* mainly on theoretic grounds, and by *Villemin* in an [illegible] discussion with *M. Leroy de Mericourt*, carried on through several months of 1874 and 1875 in the *Bulletin de l'Académie de Médecine, Paris.* The grounds on which this view is supported are chiefly the lack of constancy of any one of the usually attributed causes of the disease and their insufficiency, the history of the outbreaks and epidemics, and its analogies to some of the infectious diseases—in fact *Villemin* [illegible] it a sort of species of typhus and gives some apparently direct evidence of its propagation by contact or association. Against these views *Leroy de Mericourt* offers the usual arguments for the generally accepted notions of the disorder, agreeing with *Villemin* only in the rejection of *Garrod's* theory that the antiscorbutics are effective on account of the potash salts which they contain. The idea that scurvy is due to a specific infection or miasm has found few other supporters, though *Immermann* himself admits that the latter cannot always be excluded with absolute certainty. Nevertheless, one of the latest writers who takes up the subject, *Fabre,* adopts this view. After discussing and maintaining a neuropathic theory of purpura, he takes up scurvy in its relations with this disease, notes its analogies to it, together with its own peculiarities of origin, and comes finally to the conclusion that scurvy is a miasmatic affection which especially affects the nervous system. These two or three are exceptional among the recent writers on the etiology of scurvy, all the others holding the commonly accepted theory that it is a disease induced by defective nutrition or surroundings in one form or another. Among the more special causes, *Kirchenberger* noticed that a sudden depreciation of the quality of food and regimen seemed to produce scurvy in men accustomed to better living, while others less robust, but used to hard fare, remained well under the same circumstances. *De Lisle* offers the theory that accumulations of nitrogenous elements in the organism are the cause, and supports it by many histories of scurvy following a too nitrogenous diet with defective elimination by the skin and lungs, etc., as often is the case under the conditions in which outbreaks of scurvy occur, such as sea voyages, travelling or living in cold climates, etc. *Revillout* reports a case of scurvy in a vegetarian whose animal food consisted merely of milk and eggs, with no meat whatever. The patient, however, was obliged to undergo much fatigue and his meals were irregular and scanty. Hence *Revillout* concludes that, in estimating the causes of scurvy, the character of the diet must not alone be taken into consideration, but in connection with other circumstances, such as surroundings, habits, overwork, loss of rest, etc. This agrees with what had been already said by *Niemeyer* and others, who had also pointed out that it may occur among those whose diet is entirely vegetable. There is reason to believe also that some peculiar conditions of soil may favor the development of scurvy. Thus an outbreak of scurvy occurred in the jail at Akola, India, in 1878, which is instructive, or at least suggestive, in this regard. At the time of the outbreak the prisoners were receiving four ounces of vegetables daily, which were increased to eight ounces of potatoes, onions, carrots and yams, and yet one hundred and twenty cases occurred, with twenty deaths. Dr. *Little,* the sanitary commissioner of the Hyderabad Districts, did not attribute the epidemic to any local unsanitary conditions in the jail, for many were admitted with the disease fully developed; the disease was general in the surrounding population and many well-to-do natives, who could afford to live well, suffered. It also affected a detachment of infantry stationed at Akola, so that there was scarcely a man fit for duty, and it had to be relieved by another detachment. Dr. *Little* believed scurvy to be endemic over the whole district, but chiefly in those parts where the soil is impregnated with saline matter. "It is only, however, after seasons of drought followed by rainfall that the disease manifests itself in great intensity. The hot weather of 1877 was characterized not only by a scarcity of water, but also of food. The consequence of this scarcity was increase of crime, and the jails were filled with men in whom a previous course of bad food and bad water had engendered a habit of body predisposing them to scurvy and its allied diseases. The rainfall of 1878 was unusually heavy, so that the subsoil was saturated with water, and the level of the wells rose to within a few feet of the surface. The salt present in the soil being dissolved by the subsoil water, would naturally find its way to the wells. The injurious effects of cold and moisture in the production of scurvy are generally admitted; but, in addition, Dr. *Little* believes that large quantities of salt in the soil are an important factor in its production. This view is borne out by the civil surgeon of Akola, who arrived independently at the same conclusion after investigating another epidemic of the same disease."

The pathology of scurvy formed the subject of several papers. *Ralfe* by his examinations confirmed *Garrod's* discovery that the potash constituents of urine are decreased and comes to the conclusion that the primary lesion in the disorder is a deficient alkalinity of the blood. *Hohlbeck* thoroughly examined the urine in eight cases of scurvy. He found in the first days of the disorder a very marked decrease in quantity of the urine with increase in its specific weight, and heightened color, diminution of chlorides and proportional increase of urea and a relative decrease of potash rather than of soda, agreeing rather with *Duchek* than with *Garrod* in his results. This febrile urine was found, however, only in the first stage, later it approached more nearly the normal composition. *Uskow* made careful microscopic examinations in a number of cases, and found the lesions to consist in dilatation and choking of the capillaries with extravasations of red corpuscles and alteration of the endothelium. In severe cases, these alterations lead to gangrene of the papillæ of the gums, and destruction of the substance of the bones in parts of the body, especially in the anterior extremities of the ribs. Whether these conditions depended upon an alteration of the blood he could not say, as microscopical examination revealed no disproportion or abnormal diminution of the red and white corpuscles.

Dr. *Scheby-Buch* discusses the diagnosis of traumatic extravasations from contusions, and those in scurvy and purpura, which make their appearance spontaneously without external cause. The conclusion he reaches is, that there is nothing in the characters of the extravasations themselves that affords a certain means of diagnosis, unless the skin is broken or the instrument that produced the traumatism has left marks of its presence.

The other memoirs on our list, though of value, do not call for special notice here. The outbreak of scurvy in the British Arctic expedition of 1875 and '76 incited several articles, but can hardly be said to have materially added to the already existing ideas on the subject. The value of lime-juice a prophylactic is very strongly emphasized in the literature called out by this outbreak. The treatment of the disease and its prevention are mainly involved in the question of its etiology, and there is nothing in the recent publications on this special subject of sufficient importance to require detailed abstracts.

MORBUS MACULOSUS WERLHOFII.

(*Syn. Purpura hæmorrhagica.*)

BIBLIOGRAPHY.

Kinnicutt, F. P.: A Case of Peliosis Rheumatica. Arch. of Dermatol., I., 1875, No. 3.—Penzoldt, F.: Blutbefund bei der Werlhof'schen Krankheit. Erlanger Sitzungsber., Feb. 11th, 1878.—Gibert: Note sur deux cas de Rhumatisme Petechial. Rév. Mensuelle, Nov., 1877.—Foerster: In C. Gerhardt's Handb. der Kinderkrankheiten. Tübingen, 1878, Bd. III., i., p. 216.—Bonreif: Rec. de mem. de méd. milit. Tome XXXIV. (3e ser.), p. 172.—Davis, N. S.: Cases of Purpura hæmorrhagica with Observations on the Pathology and Treatment of the Disease. Chicago Med. Jour. and Exam., June, 1878.—Carter, M.: A Case of Purpura Hæmorrhagica. Med. and Surg. Rep., XXXIX., 1878, p. 458.—Millard, P. H.: Purpura Hæmorrhagica. Transact. Minn. Med. Soc., 1878, 23.—Widal: Purpura hémorrhagique sans Taches purpurines sur la Peau et ayant simulé une Fièvre typhoide. Mort. Autopsie. La France méd., Aug. 21st, 1878.—Bouchut: Purpura hæmorrhagica, etc. (Hôp. des enfants malad.). Gaz. des Hôp., 1878, Dec. 3d and 10th.—De Nicola: Un caso di morbo maculoso di Werlhof. Indipendente. Torino, 1878, XXIX., p. 449.—Schneider, J. R.: Ueber Purpura hæmorrhagica. Correspondenzbl. f. schweiz. Aerzte, 1878, VIII., p. 593. —Vaillard, L.: Etude sur la maladie de Werlhof. Rec. de méd. milit. Paris, 1878, XXXIV., p. 321.—Alix: Deux observations de Purpura hæmorrhagica aigu. Lyon Médical, April 7th, 1878.—Meslier: Observation pour servir à l'Histoire des complications de la maladie de Werlhof par l'albuminurie Brightique ou non Brightique. Bull. gén. de Thérap., XCV., p. 220, Sept. 15th, 1878.—Hallowes: Hemorrhage into the Cerebral Ventricles. Brit. Med. Jour., Nov. 16th, 1878, p. 733.—Claiborne, J. H.: Case of Purpura hæmorrhagica cured by Mercury. Virginia Med. Monthly, April, 1878.—Pollack, M.: Purpura rheumatica multiformis. Med. chir. Centralbl., 18, 1878.—Cavalie, F.: Purpura hémorrhagique chronique avec Paralysie alterne symptomatique. Bull. gén. de Thérap. XCVI., 1879, p. 125.—Bienecki: Morbus maculosus Werlhofii. Gaz. Lekarsk. Warszawa, 1879, XXVI., p. 251.—Duncanson, A.: Purpura hæmorrhagica. London Lancet, 1879, II., p. 33.—Graham, J. E.: A Fatal Case of Purpura Hæmorrhagica. Canad. Jour. Med. Sci., 1879, p. 326.—Karpinski, K.: K. kasuistiki Febris Intermittens, Purpura hæmorrhagica, Retinitis hæmorrhagica. Wrach. Vedomosti, St. Petersb., 1879, IV., p. 839.—Silver: A Case of Purpura Hæmorrhagica. Death. Med. Times and Gaz., 1879, II., 581.—Bouchut: Des Hemorrhagies de la Rétine dans les maladies du cerveau, dans le Typhus, dans le Diathèse hémorrhagique et dans le Purpura. Paris méd., 2d ser., V., 1879, p. 201.—Christian, J.:

Hæmatome de l'oreille et Purpura hémorrhagique chez un Paralytique général. Ann. méd. Psychol., 6 ser., III., 1879, p. 398.—DUFOUR, A. C. M.: Du Rhumatisme dit hémorrhagique et du Purpura rhumatismale. Thèse de Paris, 1879.—IGLESIAS: Purpura hæmorrhagica. Ann. R. Acad. de méd., Madrid, 1879, L, p. 143.—RIGAL: Purpura hæmorrhagica aigu dans une affection cardiaque. La France méd., April 2d, 1879, p. 214.—ROSE, W. H.: Purpura or Land Scurvy. Med. and Surg. Rep., April 19th, 1879.—HINDENLANG, C.: Pigmentinfiltration von Lymphdrüsen, Leber und anderen Organen in einem Fall von morbus maculosus Werlhofii. Virchow's Archiv, LXXIX., iii., 1880, p. 492.—SHAND: Purpura hæmorrhagica treated with Astringents and Faradization. London Lancet, July 19th, 1879.—BOZENET: Concours médical, July 12th, 1879. (Arch. of Dermatol., Oct., 1879, p. 434).—FABRE, A.: Les Relations Pathogéniques des Troubles Nerveux. Paris, 1880. Chapters VIII., IX., X., XI., on the Neuropathic Congestions and Hemorrhages, Purpura, Scurvy, etc.

Morbus maculosus Werlhofii or purpura hæmorrhagica is not a perfectly well-defined species of disease, and on this account there is some difficulty in accurately compiling its bibliography. The above list of papers, though it includes more titles than have been given by *Immermann* in either the first or the second editions, might have been made much more extensive had it comprised all the memoirs and communications that have been published within the past three or four years on simple purpura, rheumatic purpura, etc. As regards the earlier literature of these disorders, the meagreness of *Immermann's* bibliography is somewhat remarkable when we consider the comprehensiveness of his definition of the disease.

The etiology of the disorder is not elucidated to any extent by any of the writers of the articles enumerated above. Some, and among them *Bouchut*, hold to the view, discredited by *Immermann*, that, as a rule, it occurs as a result of bad surroundings, ill ventilation, insufficient and improper food. *Bouchut* even admits a deficiency of fresh vegetables in the diet as a cause, thus bringing it very near to scurvy in its causation. Several of the writers indeed consider the two as only different manifestations of the same morbid condition. Among these, *Bozenet* relates a case in which the same patient suffered from both forms of disease, as illustrating their relations and differences. A soldier, who had had well-marked scurvy seven years before, had, after a long walk through the snow, a fairly typical attack of purpura hæmorrhagica, the convalescence from which was complicated with a mild gingivitis, a symptom generally considered as diagnostic of scurvy and almost invariably lacking in purpura. According to *Fabre*, all the species or varieties of purpura are to be considered only as groups of symptoms, dependent on the nervous system and arising from a great number of widely differing morbid conditions. In this light the disease of *Werlhof* is only a special clinical form of disordered vaso-motor innervation, that may have as remote causes the rheumatic or scorbutic diathesis, some alteration of the blood from visceral disease, or anything whatever that can exert the needed influence on the vaso-motor centres. This neurotic theory of the disease appears thus to account as well for its occurrence after various severe general disorders as for its appearance spontaneously or after exposure to cold, hardships, bad sanitary conditions or miasms. *Immermann*, however, in his second edition omits the semi-favorable mention of this theory which appeared in the earlier one, preferring to offer no hypothesis whatever for the more mysterious cases that are met with occurring suddenly in individuals previously sound.

Gibert reports two cases of rheumatic purpura, one apparently dependent on malarial influence and relieved by quinine, and the other, a fatal one, noteworthy on account of the complication of nephritis occurring in its later stage, a complication which seems to be frequently observed in Great Britain, but one that has not been mentioned to any great extent in the literature of the disorder. Yet this complication was mentioned as characteristic of one of the two varieties of hemorrhagic rheumatism, recognized by *M. Constantin Paul* in a paper published in 1864, the other being the association of rheumatism with hereditary hæmophilia. This paper of *Gibert* called out one by *Meslier*, in which he says that the complication of purpura with albuminuria may be either that indeterminate and little known condition in which the albumen of the blood traverses the renal filter intact, and which seems to be connected with some altered condition of the blood, or it may be due to a simple catarrhal nephritis or to *Bright's* disease. The prognosis depends on the degree of the lesion, and is, of course, most serious in *Bright's* disease. He reports a case comparable in some respects to that of *Gibert*, but in this the patient recovered. *Bouchut* reiterates the assertion made by him in his treatise on the diseases of children, that the occurrence of purpura in the chronic diseases of infants is an invariably fatal symptom. He details two cases of hemorrhagic purpura in which retinal hemorrhages were observed during life, and in one were also verified by the autopsy. Microscopic examinations were also made of the blood, and the following are his general conclusions:

"A form of simple purpura may occur in infants, not arising from any debilitating influence, but as the result of a spontaneous alteration of the elements of the blood, consisting in diminution of the red, and relative and absolute increase of the white globules, and loss of fibrine. The globules are, in great part, deformed, they contain nuclei, and are mixed with oval bodies containing two nuclei.

"This simple purpura is characterized by miliary hemorrhages of the skin, subcutaneous ecchymoses, epistaxis and hemorrhages from mucous surfaces. Hemorrhages of the retina may be detected with the ophthalmoscope, which are absorbed and reappear without giving rise to visual trouble.

"If this purpura cause an anæmia sufficient to endanger life, transfusion must be resorted to."

The condition of the blood has also been examined microscopically by *Penzoldt*, who noticed the presence of small, sharp-contoured, clear-colored red corpuscles, and numerous abnormal small white ones with what he describes as "intermediate forms." These results are interesting as indicating a sort of attending leukæmic condition in *Werlhof's* disease, but whether this is in a causal relation or not is not yet certain, nor do we know how general or frequent is its occurrence.

Several of the reported observations are of interest on account of the peculiarity of their symptoms or complications. *Cavalie* relates a case of purpura in which epistaxis and petechiæ were accompanied with the peculiar cross-paralysis associated with lesion of the crus cerebri, a left hemiplegia with paralysis of the motor oculi on the right side. No special treatment was directed to the paralysis, but it gradually disappeared, so that after six years no trace of it remained. The interest of the case is in the occurrence of extravasation in the right crus, which the author attributes to the general hemorrhagic tendency of the disease, and its gradual absorption in accordance with the common course of these effusions. *Hallowes* reports a case of purpura in a child, ending suddenly in convulsions and death. The autopsy revealed extensive ventricular hemorrhages, and he calls attention, therefore, to this danger of purpura. *Rigal* tells of a young man who was suddenly taken with purpura seven years after an attack of rheumatism with cardiac complications, leaving behind it valvular disease. The purpura was accompanied in this patient by a subjective sensation of intense cold internally, a symptom that had not been observed before in this disorder. *Widal* gives an account of a case which, apart from its hæmorrhagic character, presented none of the usual symptoms of the disorder and which he yet diagnosed as purpura hemorrhagica. The course of the disease resembled typhoid fever: there was stupor, prostration, tympanites, and for the first few days a febrile temperature rising to about 104° F., which fell suddenly to near the normal on the fourth day, after which there was no return of the fever. The bowels were constipated. Death occurred the thirteenth day after admission to the hospital, and the post-mortem examination revealed principally softening and patches of local congestion and hemorrhages in the kidneys and general congestion of the intestines, but no ulcerations of *Peyer's* patches or other characteristic lesions of typhoid fever. The *ante-mortem* suspicion of purpura was based on the absence of pyrexia and the hemorrhages, and in accordance with this diagnosis, *M. Widal* maintains that we may have purpura without the purpurine extravasations, just as we may have measles or scarlet fever without the characteristic eruption. *Hindenlang* found, in the autopsy of a patient who died of *Werlhof's* disease, the lymphatics and the liver strongly infiltrated with pigment, the chief deposit in the latter organ being in the intra-acinous connective tissue near the branching of the vena porta. The thighs and legs were the seat of the purpuric extravasations, and the pigment deposits in the lymphatics were found along those lymphatic routes leading from the cutaneous hemorrhagic patches, such as the larger lumbar and inguinal glands of the two sides, but more especially the left, that is to say along the direct absorption routes from these parts. He enters into a rather extended discussion of these facts, which, however, are of interest here only as they throw light on some of the peculiar lesions of the disorder. They seem to show that the gradual absorption of the extravasated blood in purpura, as probably also in case of extravasations from other causes, takes place through the lymphatic system, and that this may be the explanation of certain of the visceral alterations sometimes met with in fatal cases of the disease.

The skepticism expressed by *Immermann* in regard to the medical treatment of purpura hemorrhagica is not shared by all of the more recent authors. Thus *Davis* advises a free internal use of digitalis, ergot, and iron with local application of persulphate of iron to bleeding surfaces. Other authors make similar recommendations. Dr. *Claiborne*, of Virginia, reports a case of twelve months' standing, in which other treatment by iron, etc., had failed, cured in a month's time by the use of mercury, one-twelfth of a grain of the oxymuriate three times a day after meals. He claims that it acted as a tonic and alterative, but was not led to use it on theoretical grounds, but from the advice of a brother practitioner who had had similar experience. The most striking novelty in the treatment of this disease is reported by *Shand*, who found that faradization produced good results in a case in which mineral and vegetable astringents, ergot, and tonics had all been employed without success. The patient was failing rapidly when the current was applied, the sponges being moved over the whole body and no attention paid especially to the direction of the current, and this was repeated every two hours, when the bloody evacuations from the bowels ceased, and the next day the patient was very much improved, the bleeding having almost entirely stopped. Recovery was then rapid under the use of tonics and astringents. The electricity was supposed to produce its beneficial effect in this case by improving the tone of the nervous system, facilitating the coagulation of the blood, and encouraging the capillary circulation through its general stimulant effects.

TOXICOLOGY.

BY

EDWARD S. WOOD, M.D.,

OF BOSTON,

PROFESSOR OF CHEMISTRY, HARVARD MEDICAL COLLEGE.

TOXICOLOGY.

POISONING BY IODINE AND IODIDE OF POTASSIUM.

BIBLIOGRAPHY.

BERG, F.: Archiv f. exp. Pathologie und Pharmakologie, V., 1876.—REGNARD l SIMON: Union médic., XXII., 2 sér., 1876.—HUSEMANN: Petersburg. med. tschrift, XV.—RÓZSAHEGYI: Jahresbericht über die Fortsch. der Pharmacog., arm. und Toxicologie, 1878, 564.

Berg's experiments upon animals show that the hyperæmia and inflam- tion of the kidneys is the cause of the albuminuria and suppression of ne in poisoning by free iodine, whether taken into the stomach, in- ted into the blood or into cystic cavities, as in the cases caused by the jection of Lugol's solution into ovarian cysts, of which *Velpeau* has n, according to *Husemann*, out of one hundred and thirty cases irty deaths. The urine is not merely albuminous, but also con- ins blood pigment and numerous blood-globules arranged in the form casts; after death, section of the kidneys shows the existence of umerous hemorrhages into the kidney, many of the tubules being filled ith blood. This result may also be produced by the external applica- ion of preparations containing iodine, as in the cases observed by *Reg- ard* and *Simon*, in which it was caused by the application of large mounts of tincture of iodine to the scalp in children. *Rozsahegyi* found hat the occurrence of albuminuria after the administration of iodide of otassium materially diminished the elimination of the iodide by the idneys; according to his experiments, the time required for the com- lete elimination of the iodide varied from forty-five to one hundred and rty-nine hours, this variation depending, as a rule, upon the rapidity absorption. The elimination is slower after a large dose of iodide of otassium than when the same amount is given in small divided doses. *erg* was unable to detect free iodine in the secretions, even after the jection of Lugol's solution into the blood, nor could he confirm the atement of *Rose* in regard to the elimination of iodine by the mucous embrane of the stomach, since he could not detect it, either free or mbined, if the precaution were taken to tie the œsophagus, so as to revent the entrance of saliva into the stomach.

After chronic poisoning by iodide of potassium in animals, *Rozsahegyi* sometimes found interstitial contraction of the liver and fatty degeneration of the renal epithelium.

POISONING BY BROMIDE OF POTASSIUM.

BIBLIOGRAPHY.

Krosz: Archiv f. exper. Pathol. und Pharmakol., VI., 1877.—Voisin, A.: De l'emploi du bromure de potassium. Memoire couronné par l'académie. Paris, 1875.

According to *Krosz*, bromide of potassium produces, when given to healthy men, a specific action of bromine, consisting of a mild frontal headache, loss of memory, diminution of mental faculties, a sensation of weariness, a slight lowering of the temperatnre, and the familiar modifications of the reflex excitability of the cord.

Voisin distinguishes four different forms of skin eruption produced by bromide of potassium. The first and most common occupies a place between acne simplex and acne indurata. It occurs chiefly upon the face, chest, and shoulders, after the long-continued use of small doses. The next most frequent form cannot be compared with any known skin disease; it occurs usually upon the cheek, in the form of large red patches, with jagged and raised edges, upon which acne-like pustules form; these group together, making small tumors with a very hard base, and raised above the surface three or four millimeters. In the centre of this, suppuration takes place, causing an umbilical depression, at which point there is absolute insensibility. This may last for months, and become an ulcer with an irritating secretion. The other forms are a kind of erythema nodosum, sometimes combined with œdema of the skin, occurring upon the buttocks and extremities, and a moist eczema.

POISONING BY MINERAL ACIDS.

BIBLIOGRAPHY.

Walter: Untersuchungen über die Wirkung der Säuren auf den thierischen Organismus. Diss., Dorpat, 1877.

The investigations of *Walter* upon poisoning by acids have shown that the amount of carbonic acid in the blood diminishes very much after poisoning by hydrochloric or phosphoric acid, and that the amount of carbonic acid in the blood is proportional to the amount of alkali; the ingestion of acids, therefore, diminishes the power of the blood to carry carbonic acid by neutralizing a part of the alkali, and in animals some of the symptoms which are produced, such as dyspnœa, lowering of the heart's action, collapse, etc., which cannot be explained by the direct action of the acid upon the gastric mucous membrane, are due to the neutralization of the alkali in the blood, and can be successfully combated by the injection of an alkaline solution (sodium carbonate) into the blood. This injection is preferably made into some small vein as far as possible from the heart.

POISONING BY BARIUM COMPOUNDS.

BIBLIOGRAPHY.

REICHARDT, E.: Arch. der Pharm., Mai, 1874.—SEIDEL, M.: Vierteljahrsschrift für gerichtl. Medicin, XXVII., 213.—REINCKE, J. J.: *Ibid.*, XXVIII., 248.

Several cases of poisoning by barium carbonate have been recorded within the past few years. In one of these (*Reichardt's*) the poison was taken with suicidal intent. *Reincke's* cases are interesting, in the first place, since they illustrate a new source of accidental poisoning by barium carbonate, viz., the accidental admixture of this substance with meal from which pastry was made, and also from the fact that, in addition to the usual symptoms of gastro-enteritis, there occurred paralysis, beginning in the lower limbs and gradually extending upwards, until, after the lapse of seventeen hours, only the head could be moved. The intellect, sensation, and reflex action remained intact. The sphincters were also unaffected. Death occurred in twenty-eight hours after the ingestion of the poison. Three other persons who took the same amount of the poison recovered on account of early and abundant vomiting and purging, whereas in the fatal case the symptoms were delayed several hours.

POISONING BY CHLOROFORM.

BIBLIOGRAPHY.

KNOLL: Wiener akad. Sitzungsberichte. Math.-phys. Cl., 1877.—KER: Medical Times and Gazette, 1876, I., 886.—BOEHM: Ueber Wiederbelebung nach Vergiftungen und Asphyxie. Arch. f. exp. Pathol. und Pharmakol., VIII.—REGNAULD: Étude expérimentale sur le chloroforme anesthésique. Paris, 1879. —Report on the Action of Anæsthetics to the Scientific Grants Committee of the British Medical Association. British Med. Journal, 1879, Jan. 25th and June 21st.

When chloroform is inhaled through a tracheal canula, so as to avoid the reflex action caused by the irritation of the naso-pharyngeal mucous membrane produced by the chloroform vapor, there is a quickening of the respiration and sometimes a complete cessation of inspiration, which *Knoll* considers to be due to the reflex action from those fibres of the vagus which produce inspiration; but after division of the vagus, there is a slowing of the respiration and cessation of expiration, due solely to the central action: this effect is caused also by intravenous injection of chloroform. This reflex action, from the upper as well as the lower air passages, may be considered as the safeguard against the further absorption of the dangerous vapor, although, in rare cases, this very reflex action is the cause of death after a few inhalations, by producing the cessation of the heart's action or respiration.

According to *Rendle*, in twenty London hospitals, chloroform is given about eight thousand times annually, with about three deaths (1 : 2666). *Ker* states that, during the American war, of eighty thousand cases of chloroforming, there were seven deaths (1 : 11,428).

Of twenty-eight new cases collected by *Boehm*, an autopsy showed pathological heart changes in seven, and in four of these it was stated that, during life, no abnormity could be detected by physical examination.

Boehm found that, in animals, life could be restored after the heart had ceased to beat for several minutes, by artificial respiration, and at the same time by making light compression upon the heart, so as to start the circulation; he therefore strongly recommends mechanical and electrical irritation of the heart, together with artificial respiration in the treatment of chloroform narcosis. The committee on anæsthetics, appointed by the British Medical Association, also recommend the same treatment.

POISONING BY CHLORAL HYDRATE.

BIBLIOGRAPHY.

VON MERING and MUSCULUS: Berichte der deut. chem. Gesellschaft, VIII., 662.—CHOUPPE: Gaz. hebdom., 1875.—HUSEMANN: Archiv für exp. Pathol. und Pharmakol., VI., 335.—LEVINSTEIN, E.: Vierteljahrssch. für gericht. Med. und öffent. Sanitätswesen, XX., 227.

Within a few years, quite a number of cases of fatal poisoning by chloral have been reported, almost all of which have been suicidal or accidental, and resulting from the ingestion of solutions of chloral, which have been prescribed as a hypnotic, in excessive amounts. Intravenous injection, as proposed by *Oré*, for the treatment of tetanus and other diseases, and for producing anæsthesia, has also given rise to two fatal cases of poisoning.

According to *Liebreich*, only perfectly pure chloral hydrate should be used in pharmacy, since he considers that some of the causes of fatal poisoning recorded have probably been due to impurities, such as chlorocarbonic acid, which the chloral contained. Only such chloral should be used which is crystallized in the form of well-defined rhombic tables, while those preparations which are in the form of tabular masses should be excluded from the pharmacopœia.

After the ingestion of chloral hydrate by men, a substance is found in the urine which has the properties of an acid, and which has been termed by *von Mering* and *Musculus* urochloralic acid. This acid crystallizes in colorless needles which have the composition $C_7H_{12}Cl_2O_6$. It is readily soluble in water, alcohol, and a mixture of alcohol and ether; it is insoluble in pure ether. Its solutions turn a ray of polarized light to the left.

In cases of acute chloral poisoning, death results, according to *Husemann*, from cessation of the respiration, paralysis of the heart only being produced by the application of concentrated solutions directly to the heart. This cessation of the respiration is due partly to paralysis of the respiratory centres, and partly to œdematous infiltration of the lung tissue. There is also produced a very decided lowering of the temperature; thus in *Levinstein's* case the temperature fell 6.6° C. (from 39.5° to 32.9° C.) in one-half an hour.

In regard to the treatment of chloral poisoning, the subcutaneous injection of strychnia was apparently followed by good results in *Levinstein's* case, since after the injection of 0.003 grm. of strychnia the heart's action and respiration increased, and the temperature rose 0.4° C. Afterwards, 0.002 grm. more were injected with the effect of slightly accelerating the action of the heart. The carefully conducted experimants of *Husemann*, however, show that strychnia cannot be depended on as an antidote to chloral, since it cannot prevent the slowing of the heart nor the lowering of the temperature. After the ingestion of a fatal dose of strychnia, animals can be saved by the administration of a sufficient amount of chloral to produce sleep, although the ordinary therapeutic doses are of no value. According to *Liebreich*, milk should never be given in chloral poisoning, since it would favor the formation of chloroform. In all cases the stomach pump must be used to remove the poison from the stomach, since emetics cannot be relied upon in the comatose condition.

POISONING BY CARBONIC OXIDE.

BIBLIOGRAPHY.

Boehm: Ueber Wiederbelebung nach Vergiftungen und Asphyxie. Arch. für exp. Pathol. und Pharmakol., VIII.—**Evers**: Einige Fälle von Minenkrankheit.

Deut. milit.-ärtzl. Zeitsch., 1875, und Commissar. Bericht über die Erkrankungen durch Minengase bei der Graudenzer Mineurübung, *Ibid.*, 1875.—Rochelt: Wiener med. Presse, 1875.—Huenfeld: Die Blutproben vor Gericht und das Kohlenoxyd-Blut. Leipzig, 1875.—Borzyskowski: Die chron. Kohlenoxyd-Vergiftung. Greifswald, 1877.—Poleck und Biefel: Berichte der deutsch. chem. Gesellschaft, X., 2224.—Vogel: *Ibid.*, X., 792, and XI., 235.—Grehant: Gaz. med., 1878, No. 36.

No completely satisfactory explanation of all of the symptoms of carbonic oxide poisoning can as yet be given, but it is certain that it does not act simply by producing asphyxia. Many of the severe sequelæ of carbonic oxide poisoning may, however, also be the result of simple asphyxia, as *Boehm* has shown. These sequelæ are mostly of a nervous nature, such as paralysis of the extremities, bladder and rectum, disturbances of speech and vision, mental changes, etc., and are explained by *Boehm* by the fact that the paralysis of the nervous centres disappears rapidly when produced by asphyxia of short duration, but, after long-continued asphyxia, the nervous centres regain their activity only slowly, and in the reverse order to that in which they are affected. Although the action of the heart and lungs is soon restored, several weeks may elapse before the return of the coördination of movements, sensibility, intellect, etc. In the case of *Rochelt*, the patient could not stand or walk for three weeks, and did not convalesce mentally or physically for three months.

The sapper's and miner's disease has been proved by the experiments at Graudenz to be entirely due to carbonic oxide poisoning. In these cases, of which there were 81, 7 of them being fatal, the amount of carbonic oxide in the atmosphere varied from 0.01 to 0.48 per cent.

The investigations of *Grehant* showed that, if animals were compelled to breathe an atmosphere containing from 0.07 to 0.12 per cent of carbonic oxide for one-half an hour, a sufficient amount of the gas was absorbed to render from one-quarter to one-half of the red blood-globules incapable of absorbing oxygen. The presence of sulphuretted hydrogen, even in very minute amount, with the carbonic oxide, is said to increase very much the activity of the latter upon the animal organism.

POISONING BY HYDROCYANIC ACID.

BIBLIOGRAPHY.

Hilger und Weber: Centralbl. für die med. Wissenchaften, 1877, pp. 577, 593.

These authors have investigated the spectroscopic properties of the blood as it circulates in the vessels of living animals after poisoning by hydrocyanic acid, and have found that the oxyhæmoglobin bands do not disappear, even after fatal doses of the acid have been given. Hydrocyanic acid does not, therefore, produce an absence of oxygen in the living blood, although it does diminish its amount. They also noticed that cessation of the heart's action occurred much later than that of the respiration.

POISONING BY NITROBENZOL.

BIBLIOGRAPHY.

FILEHNE: Arch. für exp. Pathol. und Pharmakol., IX., 329.

Careful experiments upon the absorption of nitrobenzol show that the so-called latent period in nitrobenzol poisoning is solely due to its slow absorption; this latent period is entirely wanting if the poison is given in such a way that absorption takes places rapidly, as, for instance, when injected subcutaneously in small amounts in several places simultaneously, or when given internally, mixed with alcoholic or oleaginous fluids. The usual slow absorption is due to the fact that nitrobenzol is not miscible with aqueous fluids, and, therefore, cannot come into intimate contact with the mucous membranes. In animals poisoned with nitrobenzol, the blood loses its power of absorbing oxygen, and spectroscopic examination shows a characteristic absorption band near the position of the acid hæmatin band, together with the oxyhæmoglobin bands. The amount of oxygen in the blood was diminished to less than one per cent, and that of carbonic acid to less than nine volume per cent. These changes in the blood cause the cyanotic color of the mucous membranes and the dyspnœa.

In the treatment, milk, fat, and spirituous liquors should be avoided, since they facilitate the absorption of the nitrobenzol.

POISONING BY CARBOLIC ACID.

BIBLIOGRAPHY.

BAUMANN: Pflüger's Archiv, XIII.—**OBERST:** Deutsch. med. Wochensch., 1878, No. 15.—**RHEINSTÄDTER:** *Ibid.*, 1878.—**SONNENBERG:** Deutsch. Zeitsch. für Chir., IX, 356.—**KÜSTER:** Von Langenbeck's Archiv, XXIII., 117.

Recent investigations on the subject of carbolic acid poisoning have shown that this acid, when it gets into the system, either by ingestion or as a surgical application, pairs with the sulphuric acid to form an entirely innocuous compound, phenylsulphuric acid, which unites with a base, and is eliminated with the urine. If a sufficient amount of carbolic acid has been absorbed, there is a complete disappearance of the alkaline sulphates from the urine, so that no reaction will be obtained upon the addition of barium chloride to the urine; this test has been used to determine the degree of poisoning, which is proportional to the amount of sulphuric acid in the urine. Upon this fact has been based the recommendation of *Baumann* to administer the alkaline sulphates for the purpose of rendering inert any excess of carbolic acid which has been absorbed.

That severe general symptoms may be produced by the absorption of carbolic acid, when applied as a surgical dressing, has been conclusively shown by numerous recent cases (*Rheinstädter*, *Sonnenberg*, *Küster*, and others). In addition to the usual symptoms mentioned by the above authors, such as headache, malaise, vomiting, and often dangerous collapse, the writer has frequently observed temporary albuminuria and casts, owing to the hyperæmia of the kidneys produced by the carbolic acid. *Küster* reports five cases of poisoning by external application in which there were cerebral symptoms, and he divides the progress of the symptoms into three stages, as follows: 1st, the dark appearance of the urine; 2d, gastric disturbances, sluggish pupil and fever; 3d, cerebral symptoms, consisting of muscular trembling and spasms.

LEAD POISONING.

BIBLIOGRAPHY.

HARNACK: Ueber die Wirkung des Bleies auf den thierischen Organismus. Archiv für exper. Pathol. und Pharmakol., IX.—ROBIERRE: Des conditions dans lesquelles le plomb est attaqué par l'eau. Comptes rendus, LXXVIII.—FORDOS: Du rôle des sels dans l'action des eaux potables sur le plomb. Comptes rendus, LXXVIII. — BOUDET: Virchow-Hirsch Jahresbericht, 1874.—ANNUSCHAT: Die Bleiausscheidung durch die Galle bei Bleivergiftung. Archiv für exper. Pathol. und Pharmakol., VII.; Die Bleiausscheidung durch den Urin bei Bleivergiftung. Ibid., X.—DOWSE: Virchow-Hirsch Jahresbericht, 1875.—FÖRSTER: Beziehungen der Allgemeinleiden u. s. w. zu Veränderungen des Sehorgans. Leipzig, 1877.—[illegible]: Recherches cliniques sur l'anesthésie. Archives générales, 1848.—HAMMOND: Diseases of the Nervous System, London, 1876.—RIEGEL: Zur Symptomatologie [illegible] der Bleikolik. Deutsch. klin. Archiv, XXI.—HILTON-FAGGE: Virchow-Hirsch Jahresbericht, 1876.—FRIEDLÄNDER: Anatomische Untersuchungen eines Falles von Bleilähmung. Virchow's Archiv, LXXV.—MELASSEZ: Virchow-Hirsch Jahresbericht, 1874.—GILBERT: Répert. de Pharmacie, XIV., 395.—DUCAMP: [illegible] d'intoxication saturnine. Annales d'hygiène, XLVIII., 307.—LEOPOLD: [illegible] Vergiftung durch Einathmen des Staubes von mit chromsauren [illegible] gefärbten Garne. Vierteljahrssch. für gericht. Medicin, XXVII., 29.—[illegible] Remarques anatomiques et cliniques sur deux points particuliers de l'intoxication saturnine chronique. Gaz. médicale, 1878, No. 32.

Recent cases of chronic lead poisoning have shown a few new sources of this disease. In one of *Gibert's* cases it was due to the habitual use of red wafers for fastening newspaper clippings together. These wafers were colored with red lead and were moistened in the mouth. The other case was caused by the ingestion of large numbers of cachous—more than a box in two days. The coating upon these cachous was found to contain 0.2 grm. of lead in each box. *Ducamp* reports quite an epidemic of lead poisoning in Paris, which was traced to a baker's shop and found to be caused by the use of old painted wood for heating the ovens. The lead carbonate in the paint was converted into oxide, which was condensed upon the oven plates, where it came in contact with the loaves of bread.

In regard to the action of water conducted through lead pipes upon the metallic lead, *Fordos* has shown that the presence in the water of the earthy (calcium and magnesium) sulphates and carbonates alone completely protects the pipe by causing upon its inner surface the deposition of an adherent coating of the insoluble sulphate or carbonate of lead, but the presence, at the same time, of the alkaline (potassium and sodium) sulphates or carbonates somewhat modifies this action and diminishes the protective power of the earthy salts.

It has been noticed that, in cases of chronic poisoning, the blue line, which forms at the junction of the teeth and gums, sometimes reappears after the administration of iodide of potassium after it has once disappeared, and without any exposure of the patient to the action of lead compounds in the mean time. This shows that the blue line is not necessarily due to the mechanical application of lead particles to the gums.

Beau has found anæsthesia to be one of the most common symptoms of chronic lead poisoning. It lasts as long as the disease lasts, disappearing only with the lead cachexia. There may be complete insensibility over the whole body, and this may extend to the mucous membrane of the throat, nose, and conjunctiva. Lead arthralgia has been observed sometimes to attack the face and scalp. *Adamkiewicz* has recently described a case of general lead paralysis which resembled that of poliomyelitis anterior, and *Renaut* has twice observed symptoms of paralysis like those of a fever; these symptoms he considers as confirming the theory that lead paralysis is a consequence of a sub-acute poliomyelitis anterior.

The cases of *Gombault* and *Friedländer* are interesting in connection with the progressive muscular atrophy which is sometimes observed, the former having found numerous degenerated nerve-fibres in the peripheral spinal nerves, and the latter having discovered the same appearances in the motor nerves, in a case in which the paralysis was limited to the upper extremities.

In lead amaurosis, ophthalmologists have found neuritis optica or atrophy of the optic nerve. In one case, the amblyopia occurred in fourteen days after exposure of the patient to the lead compound.

In respect to the theory of chronic lead poisoning, the greatest advance has been made by *Harnack's* extended investigations. He concluded from his experiments: (1) that lead affects the voluntary muscles directly by placing them in a condition of exhaustion, which is soon followed by paralysis; (2) that lead produces stimulation of certain central motor apparatus, thereby causing the peculiar spasmodic movements, which sometimes increase so as to become convulsions; (3) that lead excites certain motor apparatus located in the intestinal wall which governs the intestinal movements, thereby causing general contraction of the intestines and an increase of the peristaltic action, colic and, in animals, usually diarrhoea.

The lead colic *Harnack* considers to be due to the irritation of the intestinal ganglia by the lead. In man, the principal effect of this is the general spasmodic contraction of the intestine producing constipation, while in animals the principal effect is an increase of the peristaltic action causing diarrhoea. The peculiar pulse during an attack of colic is explained, by *Harnack*, by the general contraction of the intestine, which forces the blood to other parts of the vascular system, and thus produces an increased fulness and tension of the arteries and slowing of the pulse.

The primary cause of lead paralysis, whether it is an affection of the muscles or the nervous system, is still unsettled. In favor of the theory that it is due to a poliomyelitis anterior are the facts that it affects both upper extremities symmetrically and often suddenly, that certain muscles supplied by the affected nerve are free, and the general similarity of the paralysis to that of poliomyelitis anterior. *Harnack* considers that the primary affection is muscular, and that the degeneration of the nerves is secondary. The fact that the extensors are preferably attacked may be explained by the generally more unfavorable conditions for nutrition than the flexors.

The lead arthralgia is considered by *Harnack* to be due to the action of the lead upon the central motor apparatus. The various symptoms included in the name of encephalopathia are also due to the excitation of various nervous centres, those located in the medulla oblongata and cerebrum being attacked later than the others.

COPPER POISONING.

BIBLIOGRAPHY.

HARNACK: Wirkung der Emetica auf die quergestreiften Muskeln. Archiv für exper. Pathol. und Pharmakol., III.—BRUNTON, LAUDER, and WEST: On the emetic Action of Copper. St. Bartholomew's Hospital Reports, XII.—FELTZ und RITTER: Comptes rendus, LXXXIV.—GALIPPE: Etude toxicologique sur le cuivre et ses composés. Paris, 1875.—BOURNEVILLE et YVON: Sur un cas d'épilepsie. Comptes rendus, LXXX.—BERGERON et L'HÔTE: Sur la presence du cuivre dans l'organisme. *Ibid.*—BURCQ et DUCOM: Recherches sur l'action physiologique du cuivre. Archives de physiol. normale et pathol., XL.—BUCQUOY: Virchow-Hirsch Jahresbericht, 1874.

The questions as to the presence of copper in normal human tissues, its accumulation in the tissues when copper compounds have been taken as drugs, and the rapidity of their elimination have become quite important in the toxicology of copper compounds, and were made prominent in the "Moreau" poisoning case, in which the analyses were made by MM. *Bergeron* and *L'Hôte*, who found in the liver of one of the bodies an amount of copper corresponding to 0.119 grm. of sulphate of copper, and in that of the other, 0.084 grm. They considered that copper poisoning was the cause of death in these cases, because the symptoms were not inconsistent with copper poisoning, the post-mortem examination failed to reveal a natural cause of death, and since so large an amount of copper was detected. *Galippe* took exception to this conclusion, since much larger amounts of copper have been found in the liver when death has been due to natural causes. Thus *Bourneville* and *Yvon* found in the liver of an epileptic, who had taken during life 43 grm. of the ammonio-sulphate of copper, 0.295 grm. of metallic copper, although the patient had not taken any for three months before death. This shows that copper is eliminated very slowly from the animal economy. *Yvon* found in the liver of another person, who had during life been treated with compounds of copper, 0.236 grm. of metallic copper; *Rabuteau* found 0.239 grm., and *Galippe* found 0.310, 0.220, and 0.120 grm. in different cases.

Bergeron and *L'Hôte* consider that normally the amount of metallic copper in a human liver, when no compounds of copper have been taken during life, cannot exceed 0.003 grm. and rarely reaches 0.002 grm., although they always found it in appreciable amount, even in six fœtal livers.

Galippe considers that criminal acute poisoning is impossible on account of the horrible taste of copper compounds and their emetic effect, and that chronic poisoning never occurs, since, when small doses are given, tolerance of the system for the copper compound is established without any injurious effect upon the health. Thus he gave to patients for months at a time food which was prepared with vinegar in copper vessels and allowed to remain standing, sometimes for twenty-four hours, without producing any injury, although the verdigris was often visible in the food.

MERCURIAL POISONING.

BIBLIOGRAPHY.

BRANDIS: Behandlung der Syphilis. Berlin, 1879.—KALB: Ein Fall von lethalem Mercurialismus. Erlangen. 1876.—LUDWIG: Eine neue Methode zum Nach-

weiss des Quecksilbers in thierischen Substanzen. Stricker's Jahrbücher, 1877.—Polk und Bernadi: Virchow-Hirsch Jahresbericht, 1877.—Frommüller: Ibid., 1877.—Malcz, Sillard: *Ibid.*, 1876.—Riederer: Ueber den Nachweis des Quecksilbers im thierischen Organismus. Neues Repert. für Pharmacie, XVII.—Guntz: Virchow-Hirsch Jahresbericht, 1877.

Many cases of unusually severe and even fatal poisoning after taking calomel as a medicine have recently been reported. This result is undoubtedly due to the conversion of the calomel into corrosive sublimate, which may readily take place in the stomach during digestion, and, therefore, calomel should never be given after eating. It is also stated that long contact of calomel with sugar (in powders or pastilles) will cause this change. Any disease or condition which interferes with the elimination of mercury, such as Bright's disease, may cause exceptionally severe symptoms. *Frommüller* saw chronic mercurialismus follow the wearing of stockings which were made from old leather bottles which had formerly contained mercury.

A very interesting case, in which recovery from very severe acute corrosive sublimate poisoning was not complete for two years, is reported by *Malcz*. In this case, chronic mercurialismus with mercurial tremor developed in two weeks after taking the poison.

Quantitative analyses of the various tissues for mercury have been made by *Riederer*, who found that in animals, after taking calomel, the largest amount was contained in the liver (0.0066 per cent of the fresh tissue). Mercury has also been detected in the liver of man, when none had been taken for a year before death. *Madore* found globules of mercury in the parotid pus of a child to whom a few grains had been given, several days after the omission of the medicine.

In regard to the elimination of mercury, *Riederer* found that the kidneys eliminate it quite rapidly while the mercurial compound continues to be administered, but that this elimination quickly ceases when the drug is omitted; it may, however, according to *Guntz*, be induced again by strong salt baths or by the use of Aachen waters.

POISONING BY PREPARATIONS OF CHROMIUM.

BIBLIOGRAPHY.

Gergens: Ueber die toxische Wirkung der Chromsäure. Arch. für exper. Pathol. und Pharmakol., VI.—Leopold: Ueber tödtliche Vergiftung durch Einathmen des Staubes von mit chromsauren Bleioxyde gefärbten Garne. Vierteljahrssch. für gericht. Medicin, XXVII., 29.—Von Linstow: *Ibid.*, XX., 60.

The frequency with which chrome yellow (chromate of lead) is introduced as a pigment into confectionery and ornaments for pastry, and used for coloring children's playthings, renders the cases of chrome yellow poisoning reported by *von Linstow* and *Leopold* of great importance. In *von Linstow's* cases, two children were fatally poisoned by eating less than 0.02 grm. of chrome yellow contained in pastry ornaments. One child, aged one and three-quarters years, died in forty-eight hours, and the other, aged three and one-half years, died in five days with all of the symptoms of irritant poisoning. After death, section showed inflammation of the throat, œsophagus, stomach, and duodenum, and in one spot perforation had taken place; there was also found hyperæmia of the brain and its membranes, beginning fatty degeneration of the liver, and hyperæmia of the kidneys. In *Leopold's* cases, the poisoning was caused by inhaling dust containing chrome yellow. These patients were affected with loss of appetite, malaise, in some cases vomiting, pain in the region of the stomach and umbilicus, obstinate constipation and debility. These symptoms disappeared in a few weeks after removal of the cause, except in the case of an infant, nine weeks old, who died in six or eight days after the beginning of the symptoms. In this case, there were fever, restlessness, shrieking, several yellow fluid stools daily, redness of the skin over the chest and abdomen, parched lips, and, just before death, short respiration. After death, there was found inflammation and perforation of the stomach, as in *von Linstow's* cases. The poison could only be detected in the lungs, where 0.0036 grm. were isolated.

In animals poisoned with the chromate of potassium, *Gergens* observed great parenchymatous degeneration and hemorrhage into the kidney, with simultaneous albuminuria. He considers the cause of this to be the decomposition of the chromate, when it comes in contact with the acid urine, while being eliminated by the renal epithelium; the chromic acid thus set free causes such severe irritation that parenchymatous inflammation of the organ follows.

PHOSPHORUS POISONING.

BIBLIOGRAPHY.

FRÄNKEL: Ein Beitrag zur Lehre von der acuten Phosphorvergiftung. Berl. klin. Wochensch., XV.—AUFRECHT: Die diffuse Leberentzündung nach Phosphor. Deutsch. Arch. für klin. Med., XXIII.—STORCH: Den acute Phosphorvergiftning. Kjobenhavn, 1868.—FALCK: Stoffwechsel, etc. Archiv für exper. Pathol. und Pharmakol., VII.—FRÄNKEL: Ueber den Einfluss verminderter Sauerstoffzufuhr, etc. Virchow's Archiv, LXVI.—FISCHER and MÜLLER: Virchow-Hirsch Jahresbericht, 1876.—Also Vierteljahrssch. für gericht. Med., XXIV., 1.—v. MERING: Deutsch. Zeitsch. für prak. Med., 1875.—SELMI: Berichte der deut. chem. Gesellsch., VII., 1463.—ELVERS: Vierteljahrssch. für gericht. Med., XXV., 25. —HILGER: Archiv der Pharm., XXVI., 385.—WEYL: Arch. der Heilk., 1878, 168

Of late years considerable discussion has arisen in connection with cases of phosphorus poisoning, as to the length of time which must elapse after death before all of the phosphorus disappears from the body. The experiments made by *Fischer* and *Müller* upon guinea pigs, by poisoning them with 0.023 grm. of phosphorus, showed that this substance may remain unoxidized in the body for eight weeks after death; in these experiments phosphorous acid was detected twelve weeks after death. Dr. *v. Elvers*, however, reports a case of phosphorus poisoning in man, in which free phosphorus, to the extent of 0.094 grm., was found in the stomach and intestines twelve weeks after death.

In regard to the action of phosphorus in cases of poisoning, there is no doubt that it causes a great diminution in the process of oxidation within the body, and at the same time there is an increased decomposition of the albuminous tissues, the substances formed by this decomposition being intermediate products of oxidation between the albumen and urea, which accounts for the presence of leucin, tyrosin, and lactic acid in the urine in many cases. In one of *v. Mering's* cases, 20.5 grm. of urea were found in the urine three or four days after the poisoning, and in another case, shortly before death, lactic acid was found, but scarcely any urea. Free phosphorus has also been detected in the urine in many cases, and *Selmi* obtained phosphuretted hydrogen by treating the urine with zinc and sulphuric acid.

Where jaundice exists, both the biliary pigments and acids may be found in the urine. In one case, *Leube* detected no biliary acids, although there was deep jaundice, but in another, *Hilger* obtained the acids in crystalline form from only 500 cub. cent. of urine.

POISONING BY ARSENIC AND ARSENIURETTED HYDROGEN.

BIBLIOGRAPHY.

Hofmann und Ludwig: Arsenikvergiftung durch Fuchsin. Stricker's Jahrbücher, 1877. Gies: Experimentelle Untersuchung über den Einfluss des Arsens auf den Organismus. Arch. für exper. Pathol. und Pharmakol., VIII.—Kossel: *Ibid.*, V.—Gaethgens: Zur Kenntniss der Arsenikwirkungen. Medic. Centralblatt, 1875.—Ueber die Beschleunigung des N.-Kreislaufes durch Arsenpräparate. *Ibid.*, 1876.—Knapp: Vortrag über Arsenikesser mit Vorstellung zweier solcher. Tagblatt der Naturforscherversammlung zu Graz, 1875.—Lesser: Experimentelle Untersuchungen über den Einfluss einiger Arsenikverbindungen auf den thierischen Organismus. Virchow's Arch., LXXIV.—Lebahn: Virchow-Hirsch Jahresbericht, 1868.—Skolosuboff: Sur la localisation de l'arsenic dans les tissues, etc. Arch. de physiol. norm. et pathol., 1875.—Poncy and Livon: Recherches sur la localisation de l'arsenic dans le cerveau. Journ. de Pharm. et de Chim., 1879, 344.—E. Ludwig: Wien. Med. Blatt, 1879, II., 1151, 1175, 1239,

—**Kornfeld**: Ueber eine Vergiftung durch Fliegenpapier. Vierteljahrssch. für gericht. Med., XXII., 243.—**Hamburg**: Analyse de l'air dans les appartements tendus de papier peint arsénical. Journ. de Pharm. et de Chim., XXII., 148.—**Pinkham**: A Case of arsenical Poisoning. Trans. of the Mass. Medico-Legal Soc., I., 48.

Arseniuretted Hydrogen.—**Trost**: Vergiftung durch Arsenwasserstoff bei der technischen Gewinnung des Silbers aus Blei. Vierteljahrssch. für gericht. Med., XVIII., 269.—**Wächter**: Zur Casuistik der Arsenwasserstoffintoxicationen. *Ibid.*, XXVIII., 251.

During the last few years, there have been a large number of cases of arsenic poisoning reported, and numerous investigations upon this subject have been made. The number of possible causes of both acute and chronic poisoning has also increased very much, owing to the extended use of arsenical compounds in the arts and manufactures. The employment of white arsenic and Paris green, both in the pure state and mixed with other substances, for the purpose of destroying vermin has also given rise to numerous cases of accidental and suicidal cases of poisoning. Arsenical aniline pigments have recently played the same rôle as *Scheele's* green in poisoning by wall paper, wrapping paper, paper used for purposes of instruction in kindergarten schools, clothing and children's toys, and have also been added as a coloring matter to articles of food and drink, such as confectionery, jellies, red wines, syrups and vinegar. The use of arsenite of sodium has also given rise to cases of criminal and accidental poisoning.

Among the most important of the cases of poisoning due to these arsenical pigments are those recorded by *Hofmann* and *Ludwig*, which were caused by the use of arsenical fuchsine in the manufacture of artificial flowers. One of these cases resulted fatally, and arsenic was found in all of the tissues of the body except the bones. Of six varieties of fuchsine tested at that time, only one was found to be free from arsenic. *Reichardt* mentions one specimen of aniline red which contained 1.96 per cent of arsenic, and another which contained nearly 2.5 per cent.

When poisonous doses of arsenic have been taken, there may be a long delay in the appearance of the symptoms depending upon conditions of the stomach which control absorption. Cases quoted by *Woodman* and *Tidy* show that even in fatal cases the symptoms may not appear for from six hours to four days. In one case in which 120 grains were taken, no symptoms appeared for three days.

The length of time required for the production of the fatty degeneration of the liver and other organs in arsenic poisoning is a very important question, and one which has arisen in recent legal cases. In *Pinkham's* case, in which death occurred in forty-four hours, this degeneration had reached an extreme degree. *Casper* records a case fatal in ten hours in which fatty degeneration had commenced, and the writer met with one case fatal in seven hours in which it was noticeable in the liver. *Salkowski* found that large doses of white arsenic given to rabbits caused a fatty condition of the liver, kidneys, and heart in from twenty to twenty-eight hours.

In both acute and chronic poisoning, *Scolosuboff* found the largest mount of arsenic located in the brain and spinal cord, and thus accounts for the paralyses and other nervous symptoms so frequently seen in cases of arsenic poisoning. His analyses have been confirmed by *Poncy* and *Livon*, but *Ludwig's* results have been different, since he found the largest amount in the liver in cases of poisoning, both in man and animals. *Ludwig* also found that, in non-fatal poisoning in animals, the arsenic separated from the other organs before it did from the liver. In chronic poisoning, the elimination of the arsenic is sometimes very slow, as, for instance, in *Gaillard's* case, in which it was detected in the urine forty-five days after the last dose was taken. Whether the elimination after chronic poisoning is slower than after acute remains to be decided.

Maas found in young animals fed with arsenic the same excessive development of the compact bone tissue that *Wegener* saw after feeding with phosphorus. These results were confirmed by *Gies*. *Poncy* and *Livon* noticed that the amount of phosphoric acid in the urine increased considerably, which fact they considered to be due to the substitution of arsenic for the phosphorus in the phosphoglyceric acid.

Binz and *Schulz* have shown that arsenious acid may be converted within the system partially into arsenic acid, and *vice versa*.

ARSENIURETTED HYDROGEN POISONING.

Wächter reports four cases caused by filling toy balloons with hydrogen which contained some arseniuretted hydrogen, and *Trost* gives the history of nine cases due to the evolution of arseniuretted hydrogen in a process for separating silver from zinc by means of impure hydrochloric acid. All of the patients were jaundiced, and all had a high temperature and pulse and bloody urine. Of the thirteen cases, four proved fatal, the fatal period varying from about twenty-four hours to ten days.

POISONING BY ATROPINE.

BIBLIOGRAPHY.

RINGER and MUREL: On the Effects of Sulphate of Atropine on the Nervous System, etc. Journ. of Anat., XI., No. 2, 321.—DRAGENDORFF: Ermittelung von Giften. St. Petersburg. 1876.—BINZ: Berl. klin. Wochensch., 1878, 768.—HEUBACH: Arch. für exper. Pathol. und Pharmakol., VIII., 31.—KNAPSTEIN: Berl. klin. Wochensch., 1878, No. 27.—HUSEMANN: Arch. für exper. Pathol. und Pharmakol., VI., 443.—**Fassbender**: Berichte der deut. chem. Gesell., IX., 1357.—**Ladenburg**: *Ibid.*, XIII., 909.

The question as to the antagonistic action of atropia and morphia has given rise to a large number of recent investigations. *Binz* recognizes a limited antagonism and considers that the careful administration of morphia can diminish the dangerous excitation of the nervous centres caused by atropia. *Heubach* is of the same opinion. *Knapstein*, however, considers that the two alkaloids reinforce each other. The weight of evidence is in favor of a limited antagonism, and *von Boeck* recommends the careful use of morphia in all cases of atropia poisoning, where there is much psychical disturbance with a rapid pulse and frequent respiration.

Husemann finds that atropia antagonizes chloral, but as to whether the reverse is true or not is uncertain.

In all cases of suspected belladonna poisoning, careful examination should always be made of the contents of the stomach and intestines for the detection of the seeds, which are kidney-shaped, with a horse-shoe-shaped embryo; their color is gray, and they are about two by one and a half millimeters in size; their surface is rough. The seeds of the datura stramonium are black and much larger (four or five millimeters), and those of the hyoscyamus are grayish-brown and smaller (one or one and a half millimeters.

Fassbender has detected a bluish substance which is strongly fluorescent in all parts of the belladonna and in the extracts. The recognition of this substance may prove of great importance in the diagnosis of belladonna poisoning.

Ladenburg finds two alkaloids in the belladonna which, on account of their different specific gravity, he terms heavy and light atropia. The heavy is the ordinary atropia, and the light is identical with hyoscyamine. He also finds both of these alkaloids in the datura. The alkaloid hyoscyamine exists in the hyoscyamus niger together with an amorphous alkaloid, and duboisia, the alkaloid of the duboisia myoporoides, is identical with hyoscyamine.

DIGITALIS POISONING.

BIBLIOGRAPHY.

Bälz: Chronische Digitalisvergiftung. Archiv der Heilkunde, 1876, 468.—**Rames**: A l'étude des effets toxiques de la digitale. Gaz. des Hôp., 1876, XCV., 756.—**Jousset**: Empoisonnement par la digitale. *Ibid.*, CVIII., 858.—**Schnabl**: Die Wirkung des Digitalins auf den Blutkreislauf. Virchow-Hirsch Jahresbericht, 1875, I., 498.—**Cavazzani**: *Ibid.*, 1876, I., 419.—**Kramnik**: Moskauer pharmacolog. Arbeiten, 1876, 148.—**Köhnhorn**: Vierteljahrssch. für gericht. Med., XXIV., 278.

After taking digitalis in therapeutic doses for a long time, a sort of habit is established, somewhat similar to the opium habit, so that a patient does not feel well if the drug is omitted. This occurs especially in organic disease of the heart, and is well illustrated by the case reported by *Bälz*, in which the patient could do no work unless she took her accustomed dose of digitalis (0.3 grm. twice daily), but, if this were done, she felt perfectly well and could perform her ordinary labors.

Kramnik has shown that the rapidity of the blood current is altered by digitalis, being increased by small doses and diminished by large ones.

The active principles of digitalis withstand decomposition of the tissues for a long time, and have been detected by *Dragendorff* four months after death.

A very interesting case of chronic poisoning is reported by *Köhnhorn*, in which 13.7 grm. of powdered leaves were taken during five weeks. After fourteen days the patient had pain in the stomach, loss of appetite, headache, and ringing in the ears; the temperature was normal, but the pulse only 56. In eight days more, vomiting, gradual loss of strength, dimness of vision and anæmia appeared. Death occurred suddenly five weeks after beginning to take the drug. After death only a slight catarrh of the stomach and a dark fluid character of the blood were found.

ACONITE POISONING.

BIBLIOGRAPHY.

WRIGHT and LUFF: Pharm. Journ. and Transact., Sept., 1877, 164.—OULMONT: Bull. de l'acad. de méd., 1876, 363.—BOCHEFONTAINE and HARDY: Gazette hebdom., 1877, 285.—JONES: Brit. Med. Journ., 1877, 258.—DUIGENAM: Med. Press and Circ., 1878, 232.—ELLIOTT and STURGES: Lancet, 1878, 917.—LEWIN: Experimentelle Untersuchungen über die Wirkungen des Aconitins auf das Herz. Berlin, 1875.—MACKENZIE: Practitioner, 1878, 109, 185, 273.—BÖHM: Arch. für exper. Pathol. und Pharmakol., 1873, I., 388.—GUILINI: Experimentelle Untersuchungen über die Wirkung des Aconitins auf Nervensystem, Herz und Athmung. Erlangen, 1876.—GUÉNEAU: Bull. gén. de Thérap., XLVI., Liv. 7, 326.

C. R. A. Wright reports that there are contained in the aconitum napellus several principles:—Aconitine, $C_{26}H_{49}NO_{12}$, a powerful but difficultly crystallizable alkaloid, pseudoaconitine, $C_{33}H_{43}NO_{11}$, a less active amorphous substance with a large proportion of carbon, and also much more feeble decomposition products of aconitine and pseudoaconitine, viz., aconine and pseudoaconine. According to *Oulmont*, the root contains more alkaloid than the leaves, and, according to *Bochefontaine* and *Hardy*, the seeds are the most active.

Several cases of poisoning have recently been caused by a quack medicine called neuraline, which contains tincture of aconite, alcohol, and camphor, and which is much used in *England*. It has given rise to poisoning chiefly by being mistaken for other preparations and by being taken internally, although it is intended for external use.

Lewin, who has studied the physiological action of aconitine upon the heart, has shown that in small doses it diminishes the excitability of the motor nerves of the heart, and in large doses paralyzes them completely, and that it does not act upon the centres in the medulla, but upon the peripheral nerves. According to *Mackenzie*, who worked with English aconitine, this substance gradually diminishes the excitability of the peripheral sensory nerves and finally destroys it; the paralysis begins at the periphery, then extends to the nerve trunks, and finally involves the posterior nerve roots, but this last action occurs very late.

After death in animals, *Dragendorff* found more or less inflammation of the stomach and intestines, the mucous membrane being covered with thick tenacious mucus ; the kidneys were also inflamed.

Lewin found that animals could almost always be restored by artificial respiration, and *Böhm* recommends the subcutaneous injection of atropia.

STRYCHNINE POISONING.

BIBLIOGRAPHY.

RANKE: Virchow's Archiv, XXLV., 1.—DOMEC: Empoisonnement par la strychnine. Montpellier méd., Dec., 1877, 477.—GLISAN: Am. Journ. Med. Sci., Apr. 1878, 448.—BROWN: Lancet, 1875, 310.—BAILHACHE: Phil. Med. and Surg. Report., 1878, 412.—WILL: Edin. Med. Journ., 1875, 307.—CHARTERIS: *Ibid.*, Apr. 10th, 1875.—HUSEMANN: Arch. für exper. Pathol. und Pharmak., VI., 335, 426.—BIVINE: Phil. Med. Times, 1875, 721.—Landis: *Ibid.*, Oct. 13th, 1877.—HUSEMANN: Deutsch. med. Wochensch., 1878, No. 36, 39.—HESSLING: Ueber einige Antidote des Strychnins. Göttinger Dissert., 1877.—JOHNSON: New York Med. Record. 1875, 70.—PAUSCHINGER: Arch. für Anat. und Physiol., 1878, 401. —AUFRECHT: Deutsch. med. Wochensch., 1877, No. 38.—FALCK: Vierteljahrssch. für gericht. Med., XX., 193.—SONNENSCHEIN: Berichte der deutsch. chem. Gesellsch., XXII., 285.

The time required for the beginning of the symptoms in strychnia poisoning varies very greatly according to the rapidity of absorption ; in a large number of recorded cases the time varied from immediately after taking the poison to three hours. The fatal period also varied from a few minutes to six and a half hours. In *Ranke's* experiments with dogs, in which the animals were poisoned with 0.1 grm. strychnine, convulsions appeared on the average in eighteen minutes, and death occurred in thirty-five minutes after the poisoning. The sex and weight of the animal appeared to have no influence in these respects. After death, rigor mortis appeared on the average in fifty minutes.

Many investigations have recently been made in order to determine the best method of treatment in cases of strychnine poisoning, which has so much increased in frequency during the past few years. *Gliem* saw very good results from the injection of 0.02 grm. of apomorphia in a case of suicidal poisoning. *Brown* saw recovery follow the use of chloroform after poisoning with 0.2 grm. of strychnine, and *Bailhache*, after poisoning with from 0.3 to 0.6 grm. The subcutaneous injection of chloral hydrate was followed by recovery in a case reported by *Will*, in which poisoning was caused by from 0.25 to 0.36 grm.; good results were also seen by *Charteris* from the use of chloral. *Husemann* states that in animals five or six times the minimum fatal dose can be controlled by chloral hydrate, and even where exceedingly large doses have been taken, death is postponed for a very long time by the use of chloral. *Husemann* and *Hessling* find that bromide of potassium has no influence whatever in controlling strychnine poisoning, and that its combination with chloral is of no value.

Absorbed strychnine can be detected in largest amount in the liver even after subcutaneous injection. It can very rarely be found in the contents of the lower part of the intestines and never in the fæces. *Aufrecht* detected it in a case of attempted suicide in the vomitus ten hours after ingestion. It can also be isolated from the brain in fatal cases of poisoning.

Strychnine withstands decomposition of the animal tissues for a long time. According to *Ranke's* experiments referred to above, it could not be detected chemically 100, 130, 200, or 330 days after death in animals, although the extracts obtained from the liver and spleen gave the physiological test and had a bitter taste. In a case of human poisoning, however, the writer was able to isolate the strychnine in the form of crystals, which reacted both chemically and physiologically with all of the tests for strychnine, more than a year after death.

Frogs are best suited for the performance of the physiological test, on account of their small size and consequently small amount required to kill them, although if a sufficient amount of material can be obtained, rabbits, cats, and dogs may be used, these animals being, according to *Falck*, more susceptible to the action of strychnine in proportion to their weight than frogs.

Sonnenschein has discovered a very close relationship between strychnine and brucine, since they can readily be converted into each other artificially. By the action of oxidizing agents, strychnine can be obtained from brucine, and by the action of reducing agents, brucine can be obtained from strychnine.

OPIUM AND MORPHIA POISONING.

BIBLIOGRAPHY.

BROCHIN: Gaz. des hôp., 1877, 226.—KINKEAD: New York Med. Record, Dec., 1877.—PLANT: *Ibid.*, Nov., 1877.—SIEVEKING: Med. Press and Circ., July,

1878.- -DALBANNE: Essai sur quelques accidents produit par la morphine. Paris, 1877.—ESENBECK: Morphiumsucht. Memorabilien, 1877, II., 495.—LEVINSTEIN: Berl. klin. Wochensch., 1877, 69; 1876, 183; Die Morphiumsucht. Berlin, 1877.—DUDGEON: Edin. Med. Journ., Sept., 1877, 239.—LABORDE: Gaz. des hôp., 1877, 45.—KRAGE: UeberAlbuminurie und Glycosurie nach Morphium. Anklam, 1878. —-FIEDLER: Deutsch. Zeitsch. für prak. Med., 1874, 231, 239.—LEWIN: *Ibid.*, 1874, 241.—HIRSCHBERG: Berl. klin. Wochensch., 1877, 175.—RENAULT: De l'opium dans la médication tonique. Paris, 1876.—VIBERT: Journ. de thérap.. 1876, I., 8.—MARTIN, AIMÉ: Gaz. des hôp., 1874, 42 and 43.—PICARD and REBATEL: Gaz. méd. de Paris, 1878, 246.—PICARD: Comptes rend., LXXXVI., 1144.—FILEHNE: Arch. für exper. Pathol. und Pharmakol., X., 442 and XI., 45.—WITKOWSKI: *Ibid.*, VII., 247.—CLARK: Med. Times and Gazette, Aug., 1875, 185.—SCHWENIGER: Deutsch. med. Wochensch., 1879, No. 34.—HUSEMANN: Deutsch. Klinik, 1874, Nos. 1, 3, 6, and 10.—WOOD, H. C.: Phil. Med. Times, Dec., 1875, 145.—SEWALL: Med. Press and Circ., Apr., 1878, 275.—PALEN: New York Med. Record, Dec., 1878, 490.—GARRISON: Phil. Med. and Surg. Report., 1874, 111.—DOUGALL: Glasgow Med. Journ., Oct., 1877.—LENTE: New York Med. Record, Jan., 1874, 8.—HARWOOD: Phil. Med. and Surg. Report., 1874, 423.—WOODSWORTH: Practitioner, Oct., 1874, 251.—HEATON: Med. Times and Gaz., Apr., 1875, 413.—BURKE: Phil. Med. and Surg. Report., Dec., 1876.—WHARTON: Phil. Med. Times, 1876, 403.—HULL: *Ibid.*, 1876, 581.—MORFIT: Phil. Med. and Surg. Report., Dec., 1877, 466.—LAMADRID: Phil. Med. Times, Mar., 1878, 271.—WILSON: *Ibid.*, June, 1878, 410.—HAYNES: *Ibid.*, Sept., 1878, 577.—SMITH: Phil. Med. and Surg. Report., 1874, No. 14, 831, and No. 21, 401.—OLIVER: Amer. Journ. Med. Sciences, 1876.—BRAITHWAITE: Lancet, 1878, 874.—MARTIN: Phil. Med. Times, Jan., 1874, 231.—BUSEY: *Ibid.*, 1876, 319.—MOINET: Edin. Med. Journ., Aug., 1875, 134.—LEIDESDORF: Wien. med. Wochensch., 1876, Nos. 25 and 26, 617 and 647.—DRAGENDORFF: Ermittelung von Giften. St. Petersburg, 1876.

Among the numerous cases of opium and morphia poisoning recorded of late years, we find many illustrations of some of the rarer symptoms. Thus *Brochin* reports one, in which two and a half milligrams produced irregular respiration, temporary cessation of the heart, and deep narcosis. In *Kinkead's* case, complete consciousness appeared after the use of emetics, but stertor and myosis came on suddenly and death followed in eighteen minutes. In *Plant's* case also, a complete remission followed by sudden death occurred. Quite a number of cases of pneumonia appearing during the course of opium poisoning have also been reported.

Picard and *Rebatel* ascribe the diminution of the heart's action to paralysis of the excito-motor nerves of the heart, and *Picard* was able to detect with the microscope dilatation of the vessels in a frog. The respiratory phenomena are, according to *Filehne,* due not merely to the action of morphia in diminishing the excitability of the respiratory centre, but also to the variations in the flow of blood to the medulla oblongata produced by the action of the morphia upon the vaso-motor centre.

The order in which the various motor centres are affected by morphia has been studied by *Witkowski,* who found that those movements controlled by the centres located in the corpora quadrigemina were first attacked, then those dependent upon the cerebellum, and finally those located in the medulla oblongata.

After death in cases of acute opium poisoning the odor of the opium may be entirely absent, as in *Clark's* case, in which both morphia and meconic acid were detected chemically.

In reference to the prognosis, the condition of the respiration is of the greatest importance. If the respiration, which is becoming gradually feebler, can be increased by treatment, there is a probability that it can be restored, although in rare cases a diminution again takes place and is followed by death.

Many recent cases show the beneficial effect of the administration of strong coffee or tea, given subcutaneously if necessary. Also the value of the subcutaneous injection of atropia cannot be doubted; the principal point in connection with this treatment is in the increase of respiration which is brought about by the injection of therapeutic doses of atropia, so that in many cases life is saved by the more effective respiration thus produced, and in those cases in which death takes place, the progress of the symptoms is more favorable. The heart's action is also increased by the atropia. The atropia should be given in small doses, repeated if necessary, care being taken not to give so much that death is finally caused by the atropia, the physiological action of which lasts about twelve hours longer than that of morphia. The committee appointed by the British Medical Association report that "the beneficial action of the sulphate of atropia, after the administration of large doses of meconate of morphia, is probably due to the action which the sulphate of atropia exercises upon the blood-vessels. It causes contraction of these and thus reduces the risk of death from cerebral or spinal congestion, which is known to occur after the introduction of fatal doses of meconate of morphia. It may also assist up to a certain point, not precisely fixed in these experiments, by stimulating the action of the heart through the sympathetic, and obviating the tendency to death from deficient respiration observed after large doses of morphia."

A great many investigations have been made of late years upon the subject of chronic opium poisoning, and especially upon those cases which are due to the subcutaneous injection of morphia, which is becoming so prevalent, since the use of the subcutaneous syringe in the administration of morphia, especially among physicians. *Esenbach* and others have noticed slight œdema of the lower extremities in almost all cases of those who are subject to this latter habit. The loss of sexual power and sterility in cases of advanced opium poisoning are mentioned by *Levinstein* and *Dudgeon*. Affections of the eyes, amblyopia and myopia, have been noticed in a large number of cases, and *Laborde* has observed that morphia produces at first hyperæmia and later always anæmia of the retinal vessels.

Levinstein has paid special attention to the study of those cases caused by the injection of morphia. He distinguishes two classes of cases produced by this form of the opium habit, viz., the delirium tremens morphii and the intermittens morphica. The former has great similarity to the delirium tremens caused by alcohol, the difference between the two being the lack of the fatty degeneration and the characteristic mania of the morphia eater. The second form, called by him the the febris intermittens tertiana morphica, is distinguished from true malaria by the absence of the enlarged spleen and the failure of quinia to control the paroxysms. The long-continued use of morphia also produces a mental condition which can only be distinguished from cerebral paralysis by its disappearance on omitting the drug (*Fiedler*).

After death in those who have been in the habit of using morphia subcutaneously, *Schweniger* has found certain pathological changes in the skin in those parts selected for the application. The skin is much thickened, partly by infiltration and partly by true hypertrophy ; it also often contains abscesses, both small and large, and sometimes extensive and deep ulcers with callous edges are seen. There is almost always slight œdema of the lower extremities. In the brain, there is always marked congestion of the medulla and cortex, and fulness of the vessels in the meninges. The lungs are more or less expanded, and their tissue thick, dark, and so filled with blood in some places as to be mistaken for hemorrhagic infarction ; the vessels appear dilated, and the epithelium swollen, fattily degenerated, and containing pigment deposits. The heart is always enlarged, the walls of both ventricles thickened and the right ventricle always dilated, more rarely the left. Fatty degeneration of the heart has been noticed.

The principal treatment of chronic opium poisoning is the withdrawal of the opium, but whether this should take place gradually or suddenly authorities are not agreed. There is no doubt that, when sudden withdrawal can be borne by the patient, recovery takes place much more rapidly; but there are cases in which this cannot be done on account of the appearance of dangerous diarrhœa or collapse, which threatens the life of the patient, unless a return to the morphia is resorted to. *Levinstein* states that the appearance of the diarrhœa is a certain sign that the patient no longer uses morphia, and is not deceiving his physician. This diarrhœa usually yields to the ordinary remedies, but sometimes it does not, in which case it can only be controlled by a return to the morphia or opium in smaller doses. The collapse usually yields to stimulants. Pneumonia sometimes sets in as a consequence of the sudden withdrawal of the drug. In a case reported by *Busey*, epileptic convulsions occurred in addition to the ordinary consequences, and could only be stopped by a return to the morphia.

In cases in which very large doses have been taken habitually, it is usually impossible to carry out the sudden withdrawal treatment, but it is first necessary to gradually diminish the dose up to a certain point, when it may be taken away entirely. This is a very difficult method, since each reduction of the dose is liable to produce the same symptoms as the complete withdrawal, only to a less degree. Finally, a successful result is more liable to be attained if the patient is confined in an asylum, where the morphia mania can receive the appropriate care and treatment, which can be carried out only with great difficulty in private practice. *Levinstein* states that twenty-five per cent of those suffering with the morphia habit are incurable.

INDEX.

Abscesses, scrofulous, 759.
Acetonæmia, 772.
Acid, carbolic, subcutaneous injections of, in rheumatism, 721.
Aconite poisoning, 818.
Aconitia in neuralgia, 517.
Aconitine, its action on the heart, 818.
Acute ascending paralysis, 597.
    traumatic lesions of the spinal cord, 577.
Adenitis, arsenic in, 764.
    idiopathic, 763.
    iodine mineral waters in, 764.
Adenoma of uterus, 498.
Adynamic pneumonia, 286.
Albumen, filtration of, through glomeruli, 659.
    site of transudation of, 658.
Albuminuria, 655.
    arsenic in, 704.
    experiments into nature of, 656.
    febrile, 662.
    in amyloid degeneration, 702.
    in growing boys and men, 665.
    latent, 670.
    pathogenesis of, 660.
    physiological, 667.
    physiological, Johnson's views on, 670.
    reflex, 670.
    Runeberg's views, 661–663.
    with healthy kidneys, 668.
Alcohol in typhoid fever, directions for its use, 16.
    its influence in exciting transmission of epilepsy, 616.
Alkaline bromides in epilepsy, 625.
Amaurosis from lead-poisoning, 809.
Amblyopia, hysterical, 646.
Ambulant œdema, 682.
American resorts for consumptives, 332.
Amœba coli, 219.
Amyl nitrite in angina pectoris, 605.
    in neuralgia, 517.
Amyloid degeneration, 701.
Amyotrophic bulbar paralysis, symptoms of, 561.
    lateral sclerosis, lesions in, 589.
Anæmia, 737.
    amount of carbonic acid diminished in, 749.
    amount of fibrinogen in the blood in, 748.
    amount of urea increased in, 749.
    cerebral, 537.
    connection between organic diseases of central nervous system and, 742.
    essential, causes of, 751.
    essential pernicious, 750.
    essential, retinal hemorrhages in, 751.
    etiology of, 741.
    microscopic examination of blood in, 745.
    of spinal cord, 576.
    pathogeny of, 740.
    pernicious, leucine and tyrosin in, 752.
    pernicious, mesenteric glands in, 751.
    pernicious, treatment of, 752.
Anæsthesia of the larynx, 243.
Anasarca, 680.
    origin of, 681.
    puncture of limbs in, 705.
Anatomy of spinal cord, 567.
Anchilostomes, bibliography of, 218.
Aneurism, pathology of, 378.
    symptoms of, 378.
    treatment of, 379.
Aneurisms, multiple, 378.
Angina pectoris, 604.

contagion through food of, 297.
history of, 294.
in different genera, 295.
mortality in, 301.
nature of its poison, 298.
prevalence in man of, 300.
prophylaxis in, 302.
treatment of, 302.
virulent nature of, 302.
Antipyretic treatment of typhoid fever, 77.
Aortitis, acute, 874.
Aphasia in affections of the centrum ovale, 148.
Apomorphine subcutaneously in epilepsy, 634.
Apoplexy of spinal meninges, 573.
spinal, 577.
Arsenical injections into glands in lymphoma, 782.
Arsenic in albuminuria, 744.
in arthritis deformans, 739.
in idiopathic anemia, 764.
in malignant lymphoma, 781.
its influence on the lungs, 837.
poisoning by, 914.
poisoning, pathology of, 915.
Arseniuretted hydrogen, poisoning by, 914.
Arteries, diseases of the, 871.
diseases of the internal coat of the, 874.
diseases of the middle coat of
deforman
deforman
varieti
deforman
Arytenoid cartilag
Ascaris maritima.
Aspermatism, per
Aspirating cathet
fragments of uri
Aspiration in susp
gall-bladder, 471
Asthenic pneumon
Ataxia, locomotor,
Atelectasis, 802.
etiolog
stenos
treatm
Athetosis, 691.
bromide
centripe
double l
galvani
patholog
prognos
sympto
Atrophy of the tes
progressiv
Atropia hypodermi
hypodermi
ing, 822.
in night-sw
Atropine poisoning
Auditory nerve, aff

Bacteria, genera of, 182.
Balantidium coli, 221.
Balneo-therapy in syphilis, 184.
Balsamum antarthriticum indicum in rheumatism, 720.
Bamberger's mercuric peptone, 183.
Barium compounds, poisoning by, 803.
Bartels on muscles of the thorax, 298.
Basedow's disease, 606.
Bath, graduated, in typhoid fever, 19.
  the graduated, in acute rheumatism, 722.
Benzoate of soda inhalations in phthisis, 828.
Benzoates, internal administration of, in phthisis, 328.
Berkart on emphysema, 305.
Bethanien Hospital, Berlin, statistics of, 22.
Bigelow's operation for crushing and removing fragments of large calculi, 446.
Bile-ducts, newly-formed, 472.
Bilious typhoid of Griesinger, 28.
Black death, 38.
Bladder, angiomata of the, 445.
  diseases of the, 431.
  fibroma of the, 444.
  malformation of the, 431.
  medullary cancer of the, 442.
  papilloma of the, 443.
  power of absorption of its mucous membrane, 434.
  primary or isolated tuberculosis of, 438.
  stone in the, 445.
  tumors of the, 441.
Blennorrhagic rheumatism, 451.
Blood, alteration of, in rheumatism, 712.
  atrophy of, 740.
  changes in volume of, 739.
  corpuscles, methods of counting, 745.
  defibrinated, for rectal alimentation, 326.
  hyperalbuminosis of, 739.
  impairment of, a common cause of renal and cardiac disease, 358.
  in cerebro-spinal meningitis, analysis of, 159.
  in diabetes mellitus, 773.
  in melanuric fever, analysis of, 138.
Blood in purpura hemorrhagica, analysis of, 795.
  method of measuring volume of, 744.
  physiological function of, 738.
  regarded as an organ or tissue, 740.
Bone corpuscles, transformation into cartilage cells, 728.
  syphilis in infants, 181.
  tubercular disease of, 344.
Borax in epilepsy, 624.
Brain, diseases of the, 537.
Bright's disease, chronic, 683.
  disease, chronic, theories as to origin, 690–692.
  disease, chronic, vascular lesions in, 684.
  disease, Dickinson's views of vascular lesions in, 695.
  disease, high arterial pressure in, 697.
  disease, jaborandi in, 707.
  disease, morphia in, 705.
  disease, opiates in, 705.
  disease, pathological changes in vessels, Ewald's views, 698.
  disease, state of ganglionic centres in, 700.
  disease, treatment of, 703.
Broadbent's theory of conjugate deviation, 543.
Bromide of potassium poisoning, 802.
Brown's case of cholecystotomy, 470.
Brucine, its relation to strychnine, 820.
Bryant's case of cholecystotomy, 470.
Bubo, rheumatic, 764.
Bulbar complex of symptoms, 562.
Bursitis, syphilitic, 176.

Calomel in typhoid fever, 16.
Cancer of cervix uteri, treatment of, 500.
Cannabis indica in migraine, 603.
Carbolic acid, poisoning by, 807.
  acid subcutaneously in neuralgia, 516.
  acid subcutaneously in phthisis, 327.
Carbon bisulphate locally in cutaneous syphilides, 173.
Carbonic oxide, poisoning by, 805.
Cardiac hypertrophy, its relations to kidney disease, 688.

Catarrh, chronic nasal, 261.
  intestinal, 400.
  laryngeal, iodine and tannin in, 233.
Catching cold, 736.
Catheter, use of, in hypertrophied prostate, 435.
Cell-house of prison at Frankfort, Ky., 287.
Centrum ovale, 546.
  ovale, Nothnagel's anatomical division of, 546.
Cerebellum, lesions of the, 550.
Cerebral anæmia, 537.
  hemorrhage, 540.
  hemorrhage, conjugate deviation of head and eyes in, 542.
  hemorrhage, diminished reflex excitability in, 542.
  hyperæmia, 538.
  localization, 543.
Cerebro-spinal meningitis, 151.
  sclerosis, multiple, 584.
  shock, symptoms of, 555.
Cervix uteri, amputation of vaginal portion of, 482.
  uteri, cancer of, treatment, 500.
  uteri, hypertrophy of, treatment, 490.
  uteri, hypertrophy of vaginal portion of, 484.
  uteri, the galvano-cautery in hypertrophy of vaginal portion of, 484.
Chancre, excision of, 170.
  multiplicity of, 168.
Chancres, follicular, 168.
  multiple herpetiform, 169.
Chancroid, pathological condition of the, 167.
  relative infrequency of, 167.
Chancroids, phagedenic, treatment of, 168.
Charcot's metallo- and magneto-therapeutic experiments, 650.
Chauveau's experiments on the transmission of tuberculosis, 337.
Chest, deformity of, in pleurisy, 272.
Chloral in strychnine-poisoning, 820.
  hydrate in diphtheria, 93.
  hydrate poisoning, 804.
  hydrate poisoning, treatment of, 805.
Chloride of calcium in scrofula, 760.
Chloroform poisoning, 803.
  in strychnine poisoning, 820.
Chlorosis, its connection with hypertrophy of the heart, 350.
  nature of changes in, causing cardiac hypertrophy, 353.
Choked disk in pachymeningitis, 553.
Cholecystotomy, 469.
  expediency of the operation of, 471.
  for impacted gall-stones, 469.
Chorea, 639.
  arsenic in, 643.
  embolic origin of, 642.
  etiology and symptomatology of, 640.
  gymnastics in, 644.
  influence of seasons on, 641.
  in the dog, 642.
  pathological anatomy of, 642.
  propylamine in, 643.
  sodium salicylate in, 643.
  treatment of, 643.
Chromium preparations, poisoning by, 812.
Chyluria, 382.
  etiology of, 383.
  pathology of, 384.
  symptoms of, 384.
  treatment of, 384.
Cirrhosis, annular, 137.
  diffuse, 137.
  hypertrophic, 472.
  hypertrophic and atrophic: their histological differences, 473.
Climate, effects of, in chronic rheumatism, 726.
Climatic treatment of phthisis, 328.
Clysters, cold, in typhoid fever, 21.
Cobalt mines, their influence in causing new-growths in the lungs, 316.
Cocculus indicus in epilepsy, 625.
Cochin-China diarrhœa, 223.
Cod-liver oil in arthritis deformans, 729.
Cohnheim on tubercular disease of mesenteric glands, 343.
Cohnheim's views on tuberculosis by infection, 340.
Colchicum, action of, in acute rheumatism, 720.

Cold, slight diseases due to catching, 736.
  baths in the treatment of typhoid fever, 18.
  rubbing in dilatation of the stomach, 394.
Collagen in the initial sclerosis of syphilis, 170.
Collapse of lungs, 293.
Colpitis emphysematosa, 506.
  prognosis of, 506.
  symptoms of, 506.
  treatment of, 506.
Colpohyperplasia cystica, 506.
Colporrhaphy, anterior, 490.
  posterior, 491.
Commotio cerebri, 554.
  cerebri, pathology of, 555.
  cerebri, results of, 557.
Complications of acute articular rheumatism, 715.
Compressed air in the treatment of emphysema, 309.
Compression of spinal cord, gradual, 578.
Concentric cardiac hypertrophy, 688.
Concussion of the spinal cord, 578.
Conjugate deviation, Broadbent's theory of, 543.
Conjunctival syphilis, 177.
Consumption, pulmonary, 318.
Convulsions, uræmic, arrested by sinistro-lateral posture, 706.
Copper, ammoniacal sulphate of, in neuralgia, 516.
  poisoning, 810.
Cordons sanitaires against plague, 41.
Cornea, syphilis of, 177.
Corpora quadrigemina, lesions of, 550.
Corpulence, 752.
  anæmic form of, 753.
  plethoric form of, 753.
Corpus striatum, hemorrhages into, 548.
  striatum, lesions of, 548.
Coryza, 260.
Coto bark, tincture of, in phthisical diarrhœa, 324.
Cotoin and paracotoin in phthisical diarrhœa, 324.
Creasote for cough in phthisis, 326.
  in gangrene of the lungs, 314.
Croton-chloral in neuralgia, 516.
Croup, 77.
  and diphtheria, anatomical distinction between, 88.
Croup and diphtheria, distinction between classes of cases, 88.
  and diphtheria, their relations, 86.
Croupous pneumonia, 281.
  pneumonia, bibliography of, 281.
Curare in hydrophobia, 218.
Curtis' and Satterthwaite's views on diphtheritic poison, 83.
Cutaneous affections in diabetes mellitus, 777.
Cynara scolymus, tincture of, in acute rheumatism, 720.
Cystic transformation of the glomeruli of kidney in Bright's disease, 686.
Cystitis, 432.
  chronic, caused by lithotrity, 433.
  chronic, distention of bladder by linseed oil in, 434.
  chronic, lactic acid in, 434.
  chronic, nitrate of silver in, 433.
  chronic, quinine injections in, 433.
  chronic, spurred Indian corn in, 434.
  diphtheritica, 437.
  tuberculosa, 437.
Cysto-phosphatic deposits, 433
Cystorrhagia, cause of, 436.
Cystotomy for cystitis, 440.
Cysts of the epiglottis, 241.
  of the vocal cords, 242.

Dactylitis syphilitica, 182.
Deafness, syphilitic, 178.
Defibrinated blood in diphtheria, 94.
Deformity of chest in pleurisy, 272.
Delirium tremens morphii, 822.
Dementia paralytica, 559.
  paralytica, treatment of, 559.
Dengue, 139.
  cutaneous phenomena in, 142.
  effects of heat and moisture on, 140.
  febrile phenomena in, 143.
  joint diseases occurring in, 140.
  nervous phenomena in, 142.
  therapeutic measures in, 144.
Diabetes insipidus, diagnosis of, 780.
  insipidus, ergot in, 781.
  insipidus, etiology of, 780.

Diabetes insipidus, jaborandi and pilocarpine in, 781.
  insipidus, nitric and carbolic acids in, 781.
  insipidus, pathological anatomy of, 780.
  insipidus, symptoms of, 780.
  insipidus, treatment of, 781.
  mellitus, blood in, 773.
  mellitus, cutaneous affections in, 777.
  mellitus, diagnosis of, 776.
  mellitus, duration of, 776.
  mellitus, etiology of, 769.
  mellitus, eye affections in, 777.
  mellitus, pathological anatomy of, 770.
  mellitus, physiology of, 774.
  mellitus, symptoms of, 771.
  mellitus, treatment of, 778.
  mellitus, urine in, 771.
Diabetic coma, 772.
Diagnosis, general, of diseases of larynx, 227.
  of acute rheumatism, 716.
Diaphragm, rheumatism of, 716.
Diarrhœa of Cochin-China, 223.
Diathesis, hemorrhagic, 785.
Diet in typhoid fever, 15.
Digitalis habit, the, 818.
  poisoning, 817.
Diphtheria, 77.
  general treatment in, 94.
  germ theory of, 81.
  its dissemination by milk, 84.
  of bladder, 437.
  of pharynx, 266.
  specific contagium of, 82.
  treatment of, 91.
Diphtheritic poison, generation and means of transmission of, 84.
Diseases of the bladder, 431.
  of the female sexual organs, bibliography of, 477.
  of the intestines, physiological considerations, 400.
  of the spinal cord, general therapeutics, 572. [13.
Drains, their relation to typhoid fever,
Dull triangle in percussion, 275.
Dyspepsia, nervous, 394.
Dyspnœa, uræmic, 675.
Ear-complications of small-pox, 118.
Ear, syphilis of, 178.
Eccentric cardiac hypertrophy, 688.
Electricity, induced current in acute rheumatism, 720.
  in writer's cramp, 524.
Elevated regions recommended for phthisical patients, 329.
Ellis' curve in pleurisy, 273.
Embolic pneumonia, 294.
Emphysema, 304.
  diagnosis of, 306.
  prognosis of, 305.
  theory of the development of, 304.
  thoracentesis in, 277.
  treatment of, 307.
Encephalitis, acute, in pyæmia, 557.
  in mycotic endocarditis, 557.
  in mycotic osteomyelitis, 558.
  in ulcerative endocarditis, 557.
Endarteritis, acute, 374.
  fibrous, in Bright's disease, 685–686.
  verrucosa, 375.
Endocarditis, gonorrhœal, 725.
  in rheumatism, 712.
Endometritis, diagnosis of, 484.
  cervicis, diagnosis of, 486.
  cervicis, etiology of, 485.
  cervicis, local cauterizations in, 487.
  cervicis, symptoms of, 485.
  cervicis, treatment of, 487.
Endo-phlebitis, 380.
Endoscope of Nitze-Leiter, 444.
  value of, 454.
Enteralgia, 401.
  treatment of, 402.
Enteritis, 400.
Epidemic pneumonia of Laennec, 286.
Epididymitis in gonorrhœa, 455.
Epiglottis, cysts of, 241.
  neoplasm of the, 254.
  means for elevating the, 244.
  syphilitic disease of, 172.
Epilepsy, 614.
  alkaline bromides in, 625.
  bleeding and dieting in, 622.

Epilepsy, borax in, 624.
  chronic, influence of age on, 617.
  Cocculus indicus in, 625.
  combination of tonics and bromides in, 626.
  etiology of, 615.
  Gowers' treatment of, 624.
  hereditary transmission of, 615.
  pathology of, 619.
  pathological anatomy of, 621.
  picrotoxine hypodermically in, 625.
  symptomatology of, 618.
  traumatic, trephining in, 623.
  treatment of, 622.
  vaso-motor, doctrine of, 619.
Epistaxis, treatment of, 262.
Epstein's views upon the etiology of tuberculosis, 339.
Ergot in cerebro-spinal meningitis, 163.
Ergotine subcutaneously in hæmoptysis, [324.
Erysipelas, 145.
  connection between child-bed fever and, 146.
  treatment of, 147.
Essential pernicious anæmia, 750.
Ether and cod-liver oil in phthisis, 326.
Ethyl iodide in asthma, 309.
Eucalyptus globulus in gangrene of the lung, 314.
  in leucæmia, 427.
Exarteritis, 371.
Exophthalmic goitre, 606.
  goitre, pathogenesis of, 607.
  goitre, pathological anatomy of, 608.
  goitre, symptomatology of, 606.
  treatment of, 608.
External urethrotomy, advantages of, 454.
Extirpation of the larynx, 246.
Eye affections in diabetes mellitus, 777.
  complications and sequelæ of small-pox, 117.

Fat embolism of the lungs, 298.
  embolism of lungs, views of Wagner on its mode of production, 299.
  embolism with diabetes, 300.
Fatty emboli in lungs, detection by perosmic acid, 300.
Febrile albuminuria, 662.
Febris gastrica of Goltdammer, 22.
Fever after arsenical injections in lymphoma, 762.
  hay, 147.
  miliary, 147.
  relapsing, 25.
  typhoid, 3.
  yellow, 45.
Fibroma of the bladder, 444.
Fistulæ, uro-genital, treatment of, 507.
Flatness, line of, in pleurisy, 273.
Fluorine compounds in rickets, 110.
Follicular pharyngitis, 264.
  pharyngitis, treatment of, 265.
Fothergill's treatment of night-sweats, 825.
Fuchsine in albuminuria, 703.
Functional disease of the heart, 359.
Fungus theory, objections to the, 133.

Gall-bladder, the aspirator in suspected dilatation of, 471.
Galvano-cautery in hypertrophied prostate, 440.
  in laryngeal disease, 231.
Ganglionic centres, state of, in Bright's disease, 700.
Gangræmia, 312.
Gangrene of the lung, 312.
  of the lung, diagnosis and prognosis, 313.
  of the lung, etiology of, 312.
  of the lung, treatment of, 314.
  of the lung treated by incision, 315.
  traumatic, of lungs, 312.
Gastralgia, 399.
Gastritis, 390.
  phlegmonosa, 391.
Gelsemium in neuralgia, 516.
  in writer's cramp, 525.
General diagnosis and therapeutics of diseases of the larynx, 227.
Gerhardt on embolic pneumonia, 297.
Germ theory of diphtheria, 81.
Glanders, 187.
  accessory causes of, 187.
  duration of, 191.

Glanders, prognosis and treatment of, 192.
spontaneity of, 190.
Gleet caused by chronic prostatitis, 454.
Glottis, paralysis of adductors and abductors of the, 253.
spasm of the, 256.
Goitre, exophthalmic, 606.
Goltdammer on cold baths in typhoid fever, 22.
Gonorrhœa and its sequelæ, 447.
possible complications of, 449.
Gonorrhœal rheumatism of the heart, 449.
Gout, 730.
connection between rheumatism and, 730.
connection between skin diseases and, 730.
effect of age in. 730.
effect of sex in, 730.
saturnine, 731.
specific (Lallemand's), 732.
Ground-water, low, its relations to typhoid fever, 11.
Gummata in muscles, 176.
of the skin. 172.
Gummy tumor of larynx. 238.
Gurjun balsam in gonorrhœa. 451.
Gynæcological examination. 481.

Hæmatoblasts. 738.
Hæmatocele, extra-peritoneal. 504.
Hæmochromometric methods, 743.
Hæmatogenous jaundice. 467.
Hæmatometra, 481.
Hæmaturia. malarial. 137.
Hæmoglobin. lack of. in blood-corpuscles. 746.
reduction of, in anæmia. 738.
Hæmophilia, 785.
chlorate of potash in. 788.
coincidence of leucæmia with. 788.
etiology of. 786.
influency of pregnancy in. 786.
in infants. 787.
Hæmorrhage. cerebral. 540.
Hæmorrhagic diathesis. 785.
Hæmorrhagic infarction, method of development of, 295.
infarction, views of Cohnheim and Litten, 295.
Hay asthma, 304.
fever, 147.
Heart, affections of, in gonorrhœal rheumatism, 724.
chlorosis as a cause of hypertrophy of the, 351.
functional disease of the, 359.
hypertrophy and dilatation of, 349.
hypertrophy of, due to pregnancy, 353.
hypertrophy of, due to renal disease, 355.
idiopathic dilatation of, 358.
in pseudo-leucæmia, 413.
syphiloma of, 174.
Heidenhain's views on filtration of albumen, 664.
Hemianæsthesia and hemiplegia in lesions of the internal capsule, 549.
Hemiparaplegia spinalis, 592.
Hemiplegia spinalis, 592.
Hemiopia in cortical lesions of brain, 544.
Hepatitis, miliary granulations in parenchymatous. 136.
nodular parenchymatous. 136.
Hertz on emphysema, 305.
Hog plague. 9.
Hydatid cyst of spinal meninges, 576.
Hydatids of the prostate. 442.
Hydræmic œdema. 682.
Hydrate of chloral in hydrophobia. 212.
Hydriodic acid in emphysema. 310.
Hydrobromic acid and chloroform for cough in phthisis, 326.
Hydrochlorate of rosaniline in Bright's disease, 703.
Hydrocyanic acid. poisoning by, 806.
Hydrophobia, curare in, 213.
hydrate of chloral in, 212.
increase of, 207.
in man, 207.
lymphangitis in, 210.
pathological anatomy of, 209.
period of incubation in man, 207.
prognosis of, 212.
treatment of, 212.

Hydrophobic poison, infective qualities of, 208.
Hydropoginous vessels, 749.
Hygienic measures in the treatment of emphysema, 308.
Hyoscyamia in paralysis agitans, 634.
Hypalbuminotic hydræmia, its influence in causing dropsy, 748.
Hyperæmia, cerebral, 538.
    of lungs, 300.
    of the spinal cord and its membranes, 573.
Hyperæsthesia of the larynx, 249.
Hyperalbuminosis of the blood, 739.
Hypertrophy and dilatation of the heart, 349.
    of the heart from chlorosis, 350.
    of the heart, its connection with renal disease, 355.
    of the heart due to pregnancy, 353.
    pseudo-muscular, 612.
Hypertrophic cirrhosis, 472.
Hypnotic investigations of Heidenhain, 650.
Hypostasis, unilateral, its conjunction with bilateral croupous pneumonia, 293.
Hypostatic processes in the lungs, 292.
Hysteria, 644.
    etiology of, 645.
    in children, 647.
    pathological anatomy of, 648.
    peripheral, 646.
    symptomatology of, 646.
Hysterical amblyopia, 646.
Hystero-neuroses of the larynx, 248.

Idiopathic dilatation of the heart, 358.
Immobilization in pleurisy, 276.
Impotence an early sign of diabetes, 457.
Inanition-anæmia, 741.
Incomplete inanition, 741.
Indian plague, 38.
Inflammation, chronic traumatic, of the joints, 725.
    of the pericardium, 361.
Influenza, 147.
Insolation, 539.
    anæmia of brain in, 539.
Intermittens morphica, 822.
Internal capsule, lesions of, 549.
Internal urethrotomy in stricture, 452.
Intestinal catarrh, 400.
    parasites, 217.
Intestines, diseases of, 400.
Inversion of uterus, diagnosis of, 493.
Iodide of ethyl in asthma, 309.
    of potassium in emphysema, 309.
    of potassium poisoning, 801.
Iodine in arthritis deformans, 729.
    in laryngeal catarrh, 233.
    mineral waters in adenitis, 764.
    poisoning, 801.
Iron, tincture of the muriate, in acute naso-pharyngeal catarrh, 737.
    tincture of the muriate, in acute rheumatism, 720.
Irritation, spinal, 579.

Jaborandi in Bright's disease, 707.
    in pleurisy, 276.
Jaundice, 467.
    blood-pigment in, 467.
Jurgensen's views on croupous pneumonia, 281.

Keen's case of cholecystotomy, 470.
Kidney, primary or isolated tuberculosis of, 438.
Kidneys in pseudo-leucæmia, 412.

Labio-glosso-laryngeal paralysis, symptoms of, 561.
Lacerations of the perinæum, 507.
Lange's views upon the projected pain in neuralgia, 513.
Laparotomy in myomata, 493.
Lardacein, nature of, 701.
Laryngeal appearances in cerebro-spinal sclerosis, 252.
    catarrh, iodine and tannin in, 233.
    chorea, 250.
    crisis, 252.
    mucous membrane, inflammation of, 231.
    papilloma, 242.
    phthisis, 234.
    polypi, extra-laryngeal operation for their removal, 245.
    stenosis, 173.
Laryngismus stridulus, 232.

Laryngitis, chronic, in acute infectious diseases, 233.
  herpetic, 232.
  hypoglottica, 232.
Laryngospasmus in rickets, treatment of, 110.
Laryngostroboscopy, 229.
Laryngotomia subhyoidea vera, 244.
Laryngo-tracheal percussion, 230.
Larynx, anæsthesia of the, 243.
  extirpation of the, 246.
  general surgery of the, 254.
  gummy tumor of, 239.
  hereditary syphilis of, 239.
  hyperæsthesia of the, 248.
  hystero-neuroses of the, 249.
  leprosy of the, 242.
  lupus of the, 242.
  mucous patches in, 237.
  neoplasms within the, 240.
  neuroses of the, 247.
  rheumatic paralysis of, 716.
  syphilis of, 236.
  syphilitic ulcers in, 238.
  syphiloma of, 238.
  wounds of the, 255.
Lead amaurosis, 809.
  arthralgia, 810.
  colic, 810.
  paralysis, 810.
  poisoning, 808.
  poisoning in poliomyelitis anterior, 595.
Lenticular nucleus, lesions of, 548.
Leprosy of the larynx, 242.
Leptomeningitis spinalis, 575.
  spinalis, location of exudation in, 575.
Leucæmia, 421.
  medullary, diagnosis of, 424.
  medullary, symptomatology of, 423.
  medullary, therapeutics of, 425.
  nature of, 422.
  splenotomy in, 426.
Leucin in pernicious anæmia, 752.
Lichen urticatus of Squire, 101.
Lipoma of spinal meninges, 576.
Liquor ammoniæ in acute rheumatism, 720.
Lister's antiseptic thoracentesis in empyema, 277.
Liver in pseudo-leucæmia, 412.
Local applications in acute rheumatism, 720.
Localization in monoplegia, 544.
Locomotor ataxia, 585.
  ataxia, cutaneous reflex, 586.
  ataxia, disturbances of hearing in, 587.
  ataxia, etiology of, 585.
  ataxia, optic-nerve atrophy in, 586.
  ataxia, tendon reflex in, 586.
  ataxia, thermal baths in, 588.
Loomis on high altitudes in the treatment of phthisis, 331.
  on typho-malarial fever, 14.
Lumbricoids, bibliography of, 217.
Lungs, collapse of, 293.
  fat embolism of, 298.
  gangrene of, 312.
  hyperæmia and œdema of, 300.
  hyperæmia and œdema of, pathology and etiology of, 301.
  hypostatic processes in the, 292.
  new-growths in, 316.
  retractile force of, in pleurisy, 274.
  syphilitic deposits in, 174.
Lupus of the larynx, 242.
Lymphadenitis, idiopathic, 763.
Lymphangioma, 381.
  symptoms of, 382.
Lymphangitis in hydrophobia, 210.
Lymphatic glands in pseudo-leucæmia, 411.
Lymphatics, dilatation of, 381.
  diseases of the, 380.
  inflammation of the, 380.
  narrowing of, 381.
  occlusion of, 381.
Lymphoma, granular, 763.
  malignant, 761.
  malignant, inefficacy of operative treatment in, 761.
Lymphosarcoma, metastatic, 417.
  of lungs, 316.

Malaria, experimental basis for fungoid theory of, 134.
  hypothesis of Bouchardat in, 134.
  its influence on typhoid fever, 14.

- **Malarial** fever, loss of red blood-corpuscles in, 135.
  - disease, nature of infective cause, 132.
  - diseases, 131.
  - diseases, organized fungus in, 132.
- **Malformations** of the bladder, 431.
- **Malum** coxæ senile, 728.
- **Manometric** flame in laryngoscopy, 229.
- **Massage** in chronic rheumatism, 726.
- **Masturbation**, tonic effect of the cold sound in cases of, 458.
- **Measles**, 102.
  - bacteria of, 103.
  - constitutional lesions in, 104–5.
  - mortality of, 103.
  - nature of contagium of, 102.
- **Mechanical** irritation of urethra as a tonic remedy, 457.
- **Medulla** oblongata, diseases of, 556.
- **Medullary** cancer of the bladder, 442.
- **Megalocytes**, 747.
- **Melanuric** fever, 137.
  - fever, analysis of the blood in, 138.
  - fever, analysis of urine in, 138.
  - fever, treatment of, 138.
- **Membranous** laryngitis, its identity with laryngeal diphtheria, 91.
  - laryngitis, tracheotomy in, 95.
- **Meningeal** tuberculosis, 344.
- **Meningitis**, cerebro-spinal, 151.
  - cerebro-spinal, anatomical characters of, 159.
  - cerebro-spinal, the bromides in, 162.
  - cerebro-spinal, ergot in, 163.
  - cerebro-spinal, cause of, 152.
  - cerebro-spinal, influence of sex in, 153.
  - cerebro-spinal, non-contagiousness of, 152.
  - cerebro-spinal, pulse and temperature in, 156.
  - cerebro-spinal, the skin in, 157.
  - cerebro-spinal, the special senses in, 157.
  - cerebro-spinal, symptoms of, 154.
- Meningitis in acute articular rheumatism, 715.
  - treatment of, 161.
- Menstruation, normal, 501.
- Mercurial poisoning, 811.
- Mercuric peptone of Bamberger, 183.
- Mesenteric glands, tubercular disease of, 343.
- Metallo-therapeutics, 649.
- Metastasis in rheumatic affections, 714.
- Metritis, chronic, treatment of, 484.
- Metzquer's views upon the relation of tuberculous food to phthisis, 339.
- Micrococci in rheumatism, 713.
- Microcytes, 747.
- Microcythæmia, 738.
- Migraine, 603.
  - galvanization of sympathetic in, 604.
  - ophthalmic, 603.
- Migratory pneumonia, 291.
- Miliary fever, 147.
  - tubercle, its connection with tuberculosis, 335.
- Milk as a disseminator of diphtheria, 84.
  - contamination of, its relation to typhoid fever, 8.
- Mineral acid poisoning, 802.
- Mitral valve, relative insufficiency of the, 352.
- Monas pulmonale of Klebs, 285.
- Monoplegia, cerebral localization in, 544.
- Morbid affections of the external arterial coat, 371.
- Morbilli bullosi sive pemphigoidei, 104.
- Morbus maculosus Werlhofii, 793.
- Morphia poisoning, 820.
- Motschutkoffsky on relapsing fever, 27.
- Mountain fever, 139.
- Mucous membrane, laryngeal, inflammation of, 231.
  - patches in the larynx, 237.
- Multiple cerebro-spinal sclerosis, 584.
- Muscles, ocular, syphilitic paralysis of, 178.
- Muscular atrophy, hereditary, 611.
  - atrophy, progressive, 609.
  - syphilis, 176.
- Myalgia rheumatica, 727.
- Myelitis, 580.
  - acute, inhibitory effect of, 581.
  - chronic, 581.

- Myelitis, chronic, location of the diseased process in, 583.
  - chronic, the combined-system diseases of the cord in, 582.
  - thermal baths in, 584.
  - with acute articular rheumatism, 580.
- Myocarditis, 360.
  - complications of, 361.
- Myo-lipoma of Gowers, 613.
- Myomata, Battey's operation in, 497.
  - removal by laparotomy, 495.
- Myomotomy, 495.
  - prognosis and indications of, 496.
- Myositis, chronic, of Eulenburg, 613.

- Nasal catarrh, chronic, 261.
  - diphtheria, local treatment in, 94.
  - passages, stenosis of the, 260.
  - polypus, 262.
- Neoplasms within the larynx, 240.
- Nephritis, acute parenchymatous, in acute articular rheumatism, 714.
- Nerve-stretching, 518.
  - antiseptically, 519.
  - in epilepsy, 521.
  - in neuralgia, 519.
  - in tetanus, 521.
  - in torticollis, 521.
  - manner in which it acts, 521.
  - when indicated, 520.
- Nerves, degeneration of, 528.
- Nervous dyspepsia, 394.
  - dyspepsia, differential diagnosis of, 398.
  - dyspepsia, nature of, 395.
  - dyspepsia, treatment of, 399.
  - pneumonia, 286.
  - syphilis, 175.
- Neuralgia, 511.
  - influence of the weather on, 513.
  - new formation of dentine a cause of, 514.
  - secondary to traumatism, 514.
  - treatment of, 516.
  - varieties of, 514.
- Neurasthenia distinct from hysteria, 648.
  - spinalis, 579.
- Neuritis, 531.
- Neuritis distinguished from neuralgia, 531.
  - experimental production of, 532.
  - migrans, 531.
- Neuromata, 532.
- Neuroplastic formations, 532.
- Neuroses of the larynx, 247.
- Neurotomy in neuralgia, 518.
- New-growths in the lungs, 316.
- Night-sweats treated by sponging, 335.
- Nitrobenzole, poisoning by, 807.
- Nitroglycerin in angina pectoris, 606.
- Nose, deformities of the, methods for relief, 263.
  - diseases of the, 571.
- Nux vomica in night-sweats, 325.
- Nystagmus in pachymeningitis, 553.

- Œdema, ambulant, 682.
  - Cohnheim's and Lichtheim's experiments on pathognesis of, 680.
  - hydræmic, 682.
  - of lungs, 300.
- Oligæmia, 737.
- Oligochromæmia, 738.
- Oöphoritis, chronic, diagnosis and prognosis, 503.
  - chronic, etiology of, 502.
  - chronic, pathological anatomy of, 502.
  - chronic, symptoms of, 502.
  - chronic, treatment of, 503.
- Ophthalmic migraine, 603.
- Ophthalmoscope in cerebral hyperæmia, 539.
- Ophthalmoscopic signs of pachymeningitis, 553.
- Opiates in Bright's disease, 705.
- Opium in typhoid fever, 15.
  - poisoning, 820.
  - poisoning, chronic, treatment of, 823.
- Orchitis in typhoid fever, 453.
  - variolous, 120.
- Osteomalacia, 732.
  - analysis of urine in, 735.
  - effect of pregnancy in producing, 734.
  - effects of acids in causing, 734.

Osteomalacia, muscular spasms in, 735.
  pathology of, 733.
  solution of the bones in, 734.
Ovarian cysts, diagnosis of, 503.
Over-strain of the heart a cause of hypertrophy, 350.

Pachymeningeal hemorrhage, 552.
Pachymeningitis hemorrhagica interna, 551.
  motor disturbances in, 554.
  spinalis, 574.
Palate, diseases of the soft, 255.
Papilloma of the bladder, 443.
  of larynx, 242.
Paracentesis in pneumo-hydro-pericarditis, 367.
  in purulent pericarditis, 367.
  of pericardium, 366.
Paralyses, treatment of, 528.
Paralysis, 526.
  acute ascending, 597.
  after diphtheria, 530.
  agitans, 631.
  agitans, disseminated sclerosis of cord in, 632.
  agitans, nerve-stretching in, 633.
  agitans, pathological anatomy of, 632.
  agitans, symptomatology of, 631.
  agitans, treatment of, 633.
  condition of nerves in, 529.
  facial, nature of, 529.
  from lead-poisoning, 810.
  general, of the insane, 559.
  peripheral, frequency of, 528.
  produced by nerve-pressure, 527.
  pseudo-general, syphilitic, 175.
  spastic, 588.
Parasites, intestinal, 217.
Pathology of acute articular rheumatism, 713.
  of cardiac hypertrophy in pregnancy, 353–354.
  of cardiac hypertrophy in renal disease, 357–358.
Pathology and etiology of hyperæmia and œdema of lungs, 301.
Patellar tendon-reflex in cerebral hemorrhage, 541.
Pearl disease in cattle, 337.
Penis, atrophy of, as a result of internal urethrotomy, 464.
Pepper on emphysema, 305.
Percussion, dull triangle in, 275.
  laryngo-tracheal, 230.
  in pericarditis, 363.
Perchloride of iron in diphtheria, 92.
Periarteritis, 371.
  nodosa, 371.
Pericardial effusion, 362.
  effusion, its relation to the heart, 366.
Pericarditis and endocarditis, similarity of symptoms in, 365.
  idiopathic, 362.
Pericardium, inflammation of, 361.
Perimetritis, treatment of, 504.
Perinæum, lacerations of, 507.
Peripachymeningitis spinalis, 574.
Peritonitis a complication of gonorrhœa, 449.
Perosmic acid as test of fatty emboli in lungs, 300.
Pestis siderans, 39.
Petechiæ in pseudo-leucæmia, 415.
Pharyngitis, follicular, 264.
Pharynx, diseases of the, 264.
  tuberculosis of the, 266.
Phosphorus poisoning, 813.
Phthisis a self-limited disease, 322.
  axillary temperature in, 321.
  communicability of, 319.
  hereditary predisposition to, 320.
  its connection with brain disease, 321.
  laryngeal, 234.
  mortality from, 320.
  of larynx, tracheotomy in, 235.
  venous pulsation in, 172.
Physiology of the spinal cord, 570.
Picrotoxine hypodermically in epilepsy, 625.
  in night-sweats, 325.
Plague, the, 32.
  contagious properties of the, 38.
  epidemic of, in Asiatic Turkey, 36.

- Plague, epidemics of, in Kurdestan and Arabia, 33.
  - etiology of, 40.
  - Levantine bubonic, 38.
  - origin of, 35.
  - prophylaxis of, 40.
  - symptomatology of, 37.
  - treatment of, 40.
  - whitewashing as a prophylactic against, 40.
- Pleuro-pneumonia, epidemic of, in United States, 286.
  - of cattle, 291.
- Pleurisy, deformity of chest in, 272.
  - elevation of temperature in, 272.
  - Ellis' curve in, 274.
  - immobilization in, 276.
  - jaborandi in, 276.
  - line of flatness in, 273.
  - retractile force of lungs in, 274.
  - thoracentesis in, 276.
  - treatment of, 276.
- Pneumatic treatment of emphysema, 308.
- Pneumo-enteritis, infectious, of hogs, 9.
- Pneumogastric nerve, its relation to pneumonia, 531.
- Pneumonia, contagious form of, 289.
  - croupous, 281.
  - croupous, clinical history of, 283.
  - croupous, treatment of, 283.
  - doctrine of the infectious nature of, 281.
  - embolic, 284–294.
  - embolic, pathogenesis and etiology of, 295.
  - embolic, symptomatology and diagnosis of, 297.
  - embolic, treatment of, 298.
  - epidemic, 286.
  - epidemic of, in Akerhus prison, 288.
  - epidemic of, in Frankfort prison, 286.
  - epidemic of, in House of Correction, Moringen, 288.
  - etiology of, 282.
  - fibrous, its relation to neuritis of pneumogastric, 531.
  - from sewer-gas, 285.
  - interstitial, 285.
  - migratory, 291.
- Pneumonia, morbid anatomy of, 282.
  - mortality curve of, 284.
  - percentage of, among all diseases, 290.
  - pythogenic, in Dublin, 290.
  - quinine in, 283.
  - salicylic acid compounds in, 283.
- Pneumonic fever of Flint, 282.
- Pneumopericardial friction sounds in pleurisy, 273.
- Poikilocythæmia, 738.
- Poisoning by aconite, 813.
  - by arsenic and arseniuretted hydrogen, 814.
  - by atropine, 816.
  - by barium compounds, 803.
  - by bromide of potassium, 802.
  - by carbolic acid, 807.
  - by carbonic oxide, 805.
  - by chloral hydrate, 804.
  - by chloroform, 803.
  - by chromium preparations, 812.
  - by copper, 810.
  - by digitalis, 817.
  - by hydrocyanic acid, 806.
  - by iodine and iodide of potassium, 801.
  - by lead, 808.
  - by mercury, 811.
  - by mineral acids, 802.
  - by nitrobenzole, 807.
  - by opium and morphia, 820.
  - by phosphorus, 813.
  - by strychnine, 819.
- Poliomyelitis anterior acuta, 593.
  - anterior subacuta et chronica, 594.
  - anterior chronica, fibrillary muscular contractions in, 596.
  - anterior, lead-poisoning in, 595.
  - anterior subacuta, pathology of, 595.
- Polypus, nasal, 262.
- Ponfick's views of acute miliary tuberculosis, 344.
- Post-hemiplegic disorders of movement, 635.
- Potash compounds in epilepsy, 623.
- Potassium bromide poisoning, 802.

Potassium iodide poisoning, 801.
Powers' experiments into the dissemination of diphtheria by milk, 84.
Pregnancy as a cause of cardiac hypertrophy, 353.
  nature of its influence in causing cardiac hypertrophy, 354.
Priapism in leukæmia, 463.
  persistent, 462.
Processes, hypostatic, in the lungs, 292.
Progressive muscular atrophy, 609.
  muscular atrophy, lesions of the spinal cord in, 611.
  muscular atrophy, pathological anatomy and pathogenesis of, 609.
Prophylaxis in peripheral thrombosis, 298.
Prostate gland, galvano-cautery in hypertrophy of, 440.
  gland, hydatids of the, 442.
  gland, primary or isolated tuberculosis of, 439.
  gland, proper treatment of hypertrophied, 435.
  hypertrophied, its relations to the bladder, 433.
Pruritus vulvæ, 508.
Pseudo-leucæmia, 409.
  course and prognosis of, 419.
  diagnosis of, 416.
  pathological anatomy of, 410.
  splenic injections in, 420.
  symptomatology of, 413.
  therapeutics of, 420.
Pseudo-leucocythæmia, medullary, 747.
Pseudo-muscular hypertrophy, 612.
  hypertrophy, pathology of, 613.
Purpura hemorrhagica, 794.
  hemorrhagica, etiology of, 794.
  hemorrhagica, faradization in, 797.
  hemorrhagica, treatment of, 797.
Pulmonary consumption, 318.
  consumption, diagnosis of, 321.
  consumption, general treatment of, 326.
  consumption, prognosis in, 322.
  consumption, treatment of, 324.
Pulse-curves in myocarditis, 361.
Pyæmia, acute, encephalitis in, 557.
  in small-pox, 120.
Pyrogenous substance in fever, 736.
Pythogenic pneumonia, 290.

Quarantine against the plague, 41.
Quebracho in dyspnœa, 310.
  in emphysema and bronchitis, 311.
Quinine in acute naso-pharyngeal catarrh, 737.
  in acute rheumatism, 720.
  in croupous pneumonia, 283.
  in diphtheria, 93.
  in typhoid fever, 17.
  in yellow fever, 70.

Rabies and hydrophobia, 207.
  official publication of information relating to, 211.
Race, influence of, in rheumatism, 712.
Rachitis, congenital, 106.
  fœtal, 106.
  fœtal, symptoms of, 109.
Rectal alimentation by defibrinated blood, 327.
  stenosis, 173.
Rectum, syphilis of, 173.
Relapsing fever, 25.
  fever, bacteria in, 26.
  fever, history of, 31.
  fever, reinfection, 27.
  fever, successful inoculation of blood in, 27.
Renal cirrhosis, its relation to cardiac hypertrophy, 689.
  disease as a cause of cardiac hypertrophy, 355.
  disease as a cause of cardiac hypertrophy, nature of the process, 356.
Respirator, antiseptic, in gangrene of the lung, 314.
Results of acute rheumatism, 717.
Retinal hemorrhages in essential anæmia, 751.

- Rhabditis stercoralis, 221.
- Rheumarthritis, treatment of, 712.
- Rheumatism, acute articular, acute parenchymatous nephritis in, 714.
  - acute, diagnosis of, 716.
  - electro-cutaneous sensibility in acute articular, 714.
  - acute articular, meningitis in, 715.
  - acute articular, pathology of, 713.
  - acute, results of, 716.
  - acute, salicylate of ammonia in, 719.
  - alteration of blood in, 712.
  - complications of, 712.
  - endocarditis in, 713.
  - gonorrhœal, 723.
  - gonorrhœal, solely a complication or urethritis, 724.
  - influence of race in, 712.
  - micrococci in, 713.
  - of the diaphragm, 716.
  - sequelæ of acute, 717.
  - treatment of acute, 717.
- Rheumatic affections, metastasis in, 714.
  - bubo, 764.
  - inflammation, chronic, of the joints, 725.
- Rickets, 105.
  - causation of, 107.
  - pathogenesis of, 107.
  - symptomatology of, 108.
  - treatment of, 110.

- Salicin in acute rheumatism, 717.
- Salicylate of ammonia in acute rheumatism, 719.
  - of soda in typhoid fever, 17.
- Salicylic acid a specific in acute rheumatism, 718.
  - acid as an anti-diphtheritic, 83.
  - acid in gangrene of the lung, 314
  - acid compounds in croupous pneumonia, 283.
  - acid in gout, 732.
  - acid in rheumatism, 712.
- Sanitary condition of towns in China, 11.
- Sapper's and miner's disease, 806.
- Sarcoma of spinal meninges, 576.
- Sarcosin in gout, 732.
- Schistomycetes of Letzerich, 10.
- Sciatic nerve, experiments upon strength of, 520.
- Sclera, syphilis of, 177.
- Sclerosis, multiple cerebro-spinal, 584.
- Sclerotic coat of the eye inflamed in gonorrhœal rheumatism, 724.
- Scorbutus, 789.
- Scrofula, air baths in, 760.
  - chloride of calcium in, 760.
  - cutaneous eruptions in, 759.
  - diseases of bones in, 759.
  - infectious and non-infectious, 757.
  - inflammation of mucous membranes in, 759.
  - sea-bathing in, 760.
- Scrofulosis, 753.
  - Buhl's theory of infection, 753.
  - granulation tissue at the basis of scrofulous abscesses, 758.
  - theories of its causation, 756.
- Scrofulous processes, distinction between tuberculous processes and, 757.
- Scurvy, 789.
  - etiology of, 790.
  - influence of soil in causing, 791.
  - pathology of, 792.
- Sea-bathing in scrofula, 760.
- Secondary degenerations of the spinal cord, 598.
- Seminal vesicles, cystic development of, 463.
- Senator on the connection between phthisis and brain-disease, 321.
- Sensibility, electrocutaneous in acute articular rheumatism, 714.
- Septum nasi, diseases of, 263.
- Sequelæ of acute rheumatism, 717.
- Serratus magnus, paralysis of, 530.
- Sewer-gas pneumonia, 285.
- Sewers, their relation to typhoid fever, 13.
- Skin, pathological change in, produced by subcutaneous injections of morphine, 823.
- Small-pox and vaccination, 115.
  - affections of abdominal organs in, 119.

l-pox, affections of female genital organs in, 120.
  affections of locomotor apparatus in, 118.
  catarrhal pneumonia in, 118.
  changes in liver, kidneys, and spleen in, 125.
  condition of the bile in, 125.
  condition of the internal organs in, 124.
  croupous and hypostatic pneumonia in, 118.
  diphtheria in, 119.
  ear complications in, 118.
  extravasations of blood in, 126.
  eye complications and sequelæ in, 117.
  flabby heart in, 126.
  hemorrhages from uro-genital system in, 127.
  hemorrhagic pocks in, 123.
  incrustation process in, 123.
  inflammation of salivary glands in, 119.
  pathological anatomy of, 120.
  pericarditis in, 119.
  pleurisy in, 119.
  pustules in the digestive tract in, 124.
  pustules in the trachea in, 124.
  pustules of vulva and vagina in, 125.
  pyæmia in, 120.
  secondary umbilication in, 122.
  severe affections of the larynx in, 119.
  treatment and prophylaxis, 127.
  ulcerations of nasal mucous membrane in, 118.
  umbilication in, 122.
m and tetany, 520.
  of the urethra, 453.
tic paralysis, 588.
  paralysis, chloride of gold in, 592.
  paralysis, contracture in, 590.
  paralysis, dry cups and purgatives in, 592.
  paralysis, hydromyelus of cervical cord in, 590.
  paralysis, lesions of the cauda equina in, 590.
Spastic paralysis, relaxation of the limbs in, 592.
  paralysis, treatment and prognosis, 592.
Spleen, diseases of, 402.
  diseases of, anatomical and physiological remarks, 402.
  experiments on, to determine functions of, 406.
  functions of, 405.
Splenic tumors, chronic, 409.
Splenotomy, 408.
Spina bifida, 599.
Spinal apoplexy, 577.
  cord, acute traumatic lesions of, 577.
  cord, anæmia of, 576.
  cord, anatomy of, 567.
  cord, concussion of, 578.
  cord, general therapeutics of diseases of the, 572.
  cord, gradual compression of, 578.
  cord and its membranes, hyperæmia of, 573.
  cord, physiology of, 570.
  cord, secondary degenerations of, 598.
  cord, tumors of, 598.
  irritation, 579.
  meninges, apoplexy of, 573.
  meninges, tumors of the, 576.
  myosis in ataxia, 586.
Spirillum fever of Carter, 29.
  fever, its inoculation in monkeys, 30.
Spirochæte in relapsing fever, 27.
  obermeieri, 9.
  their relation to changes of temperature, 32.
Stenosis, laryngeal, 173.
  of the nasal passages, 260.
  of the uterus, 482.
  of the uterus, operations for, 482.
  of uterus, wedge-shaped excision of lips of os in, 483.
  rectal, 173.
Sterility in the male, 456.
Stethograph in the diagnosis of emphysema, 307.
Stomach, 389.
  dilatation of, 392.

- Stomach, preliminary physiological remarks, 389.
- Stone in the bladder, 445.
- Stricture, internal urethrotomy in, 452.
- Strychnia in night-sweats, 325.
  - nitrate of, in writer's cramp, 525.
- Strychnine poisoning, 819.
- Sulphur in diphtheria, 93.
- Syphilis, 167.
  - blood-counting in, 170.
  - constitutional, nature of secretions in, 170.
  - cutaneous hemorrhage, 173.
  - distinct in its identity, 171.
  - galloping, 178.
  - hereditary, views on, 177.
  - hereditary of the larynx, 239.
  - inherited, cutaneous manifestations of, 180.
  - inherited, of mucous membranes, 181.
  - initial sclerosis of, histology of, 169.
  - its relation to marriage, 179.
  - its transmission by inheritance, 179.
  - muscular, 176.
  - of bones in infants, 181.
  - of conjunctiva, 177.
  - of rectum, 173.
  - of the larynx, 236.
  - of nervous system, 175.
  - primary, treatment of, 183.
  - prognosis of, 171.
  - tonic treatment of, 184.
  - visceral complications in, 175.
- Syphilitic cutaneous diseases, nomenclature of, 171.
  - deposits in the lungs, 174.
  - diseases of the arteries, 377.
  - parents, their relation to offspring, 179.
- Syphiloma, ano-rectal, 173.
  - of heart, 174.
  - of larynx, 238.
- Sympexions a cause of aspermatism, 463.
- Syringomyelitis, 599.

- Tait's case of cholecystotomy, 470.
- Tappeiner's experiments upon the infectiousness of tuberculosis, 336.
- Tapping phthisical cavities, 326.
- Taube's observations on an epidemic of measles, 104.
- Tayuya in syphilis, 184.
  - locally in gonorrhœa, 451.
- Temperature important in the treatment of phthisis, 330.
  - in uræmia, 676.
- Tetanus, 624.
  - calabar bean in, 630.
  - chloral in, 630.
  - chloroform in, 630.
  - etiology and symptomatology of, 627.
  - nerve-stretching in, 630.
  - pathological anatomy of, 628.
  - pigmentary degeneration of spinal ganglion-cells in, 628.
  - potassic bromide in, 630.
  - treatment of, 630.
- Tetany, 522.
- Thalamus opticus, lesions of the, 549.
- Therapeutics, general, of larynx, 227.
- Thermal baths in myelitis, 584.
- Thermo-cautery in laryngeal disease, 231.
- Thoracentesis in pleurisy, 276.
- Thyrotomy, explorative, 255.
- Tonic contractions of voluntary muscles, 600.
- Tonsils, hypertrophied, 264.
- Toxicology, 801.
- Tracheotomy in croup and diphtheria, indications for, 96.
  - in membranous laryngitis, 95.
  - value of, in ulcerative phthisis of the larynx, 235.
- Treatment of acute rheumatism, 717.
  - of chronic rheumatism, 726.
- Trichinosis, 214.
  - frequency of, 215.
  - œdema in, 215.
  - on board the Cornwall, 216.
  - treatment of, 216.
- Trigeminal, paralysis of, experiments with, 529.
- Tubercles in phthisis of the larynx, 235.
  - in the pig, 338.
- Tubercular disease of bone, 344.
- Tuberculosis, 334.
  - of the bladder, 437.

- berculosis in dogs caused by inhaling phthisical sputa, 755.
  - excited by a specific infection, 754.
  - heredity of, 345.
  - infectious nature of, 335.
  - its relations to scrofula and phthisis, 334.
  - meningeal, 344.
  - of the pharynx, 266.
  - propagated by a specific process of infection, 340.
  - similarity of many animal diseases to, 337.
  - uro-genital, 343.
- berculous disease of spermatic cord, 60.
- mors of the bladder, 441.
  - of pelvic peritoneum and cellular tissue, 505.
  - of the spinal cord, 598.
  - of the spinal meninges, 576.
  - of spleen, 409.
- phoid fever, 8.
  - fever a schistomycosis, 10.
  - fever, epidemic of, at Ascot, 8.
  - fever, epidemic of, at Kloten, 7.
  - fever, etiology of, 7.
  - fever, influence of age upon, 13.
  - fever, influence of seasons upon, 12.
  - fever, its transmission from animals, 8.
  - fever, its transmission from man to animals, 10.
  - fever, nature of, 6.
  - fever, origin of, 5.
  - fever, prevention of relapses in, 22.
  - fever, treatment of, 15.
  - pneumonia, 286.
- rosin in pernicious anæmia, 752.

- ers, syphilitic, in larynx, 237.
- ar nerve, paralysis of, 530.
- æmia, 672.
  - blood changes in, 674.
  - opium in, 706.
  - punctiform cerebral hemorrhages in, 672.
  - red blood-corpuscles in, 674.
  - temperature in, 676.
- Uræmic convulsions, pathogenesis of, 673.
  - convulsions, sinistro-lateral posture in, 706.
  - dyspnœa, 675.
- Urethra, spasm of, 453.
- Urethritis a cause of aspermatism, 461.
- Urethrotomy, external, advantages of, 454.
- Urinary casts, the chemistry of, 676.
  - casts, nature of, 677.
  - casts, varieties of, 678.
  - fibrous cylinders, origin of, 679.
  - phthisis, 438.
- Urine, analysis of, in acute rheumatism, 721.
  - analysis of, in essential anæmia, 751.
  - analysis of, in gout, 731.
  - analysis of, in osteomalacia, 735.
  - in diabetes mellitus, 771.
  - in melanuric fever, analysis of, 138.
  - in scurvy, analysis of, 792.
- Urobiline, its relation to hæmoglobine, 468.
  - jaundice, 468.
- Urobilinuria, its relation to extravasated blood, 468.
- Uro-genital fistulæ, treatment of, 507.
  - tuberculosis, 343.
- Uspensky's views of neuralgia, 512.
- Uterine adenoma, 498.
  - adenoma, diagnosis of, 499.
  - adenoma, etiology of, 498.
  - adenoma, prognosis of, 499.
  - adenoma, symptoms of, 499.
  - adenoma, treatment of, 499.
  - fibroids, etiology of, 493.
  - fibroids, treatment of, 493.
  - infarction, treatment of, 484.
  - leiomyomata, etiology of, 493.
  - myomata, etiology of, 493.
- Uterus, anteflexion of, 490.
  - anteversion of, 490.
  - changes in attitude and shape of, 488.
  - inversion of, diagnosis, 493.
  - retroflexion of, 490.
  - retroversion of, 490.
  - stenosis of the, 482.
  - treatment of prolapse of, 490.

Vaccination, advantages of humanized lymph, 128.
  and small-pox, 115.
Vaccinal syphilis, bibliography of, 116.
Vagina, treatment of prolapse of, 490.
Vaginismus, 507.
Vaginitis, pathological anatomy of, 505.
Vagus, experimental physiology of, 530.
Varicella, 99.
  inoculation of, 100.
  its relation to variola, 99.
  prurigo of Hutchinson, 101.
Variola of respiratory tracts, 124.
  vera of mucous membranes, 124.
Variolous orchitis, 120.
Veins, dilatation of the, 380.
  diseases of the, 380.
Verneuil on secondary neuralgia, 514.
Virchow's experiments upon pearl-disease in man, 337.
Visceral complications of syphilis, 175.
Visible vibration of vocal cords, 229.
Vocal cords, cysts of the, 242.
  cords, phlyctenular inflammation of, 232.
  cords, stammering of, 252.
  cords, visible vibration of, 229.
Voluntary muscles, tonic contractions of, 600.

Warm bath in treatment of chancroids, 168.
Water-traps and closets, their relation to typhoid fever, 13.
Water, use of, internally in typhoid fever, 15.
Weigert's views as to the formation of the pock-marks in small-pox, 121.
Wernicke's auditory disturbance in br
  disease, 546.
Woodward's typho-malarial fever, 14.
Writer's cramp and allied affections, 5
  cramp, electricity in, 524.
  cramp, nature of, 524.
Writing power. impaired, varieties
  523.

Yellow fever, 45.
  fever, clinical history, 59.
  fever, complications with pal
    ism, 61.
  fever, depopulation in, 59
  fever, etiology of, 46.
  fever, excretions in, 63.
  fever, filth as a factor in, 55.
  fever, heat as a factor in, 55.
  fever, history of, 46.
  fever, is it a miasmatic-contag
    disease, 55.
  fever, moisture as a factor in,
  fever, outside of the United St
    56.
  fever, pathology of, 64.
  fever, prophylaxis in, 58.
  fever, quarantine in, 58.
  fever, quinine in, 70.
  fever, sanitation in, 58.
  fever, stimulants in, 71.
  fever, temperature in, 63.
  fever, the heart in, 67.
  fever, the kidneys in, 68.
  fever, the liver in, 68.
  fever, the stomach in, 68.
  fever, tinct. ferri sesquichl.
    72.
  fever, treatment of, 69.

This book should be returned on or bef
the date last stamped below.

25M-[illegible]-50-88267

L41
Z66
1881
Cyclopaedia of the Practice
Ziemssen

L41
Z66
1881
Cyclopaedia of the
Practice of Medicine
Ziemssen Supplement

NAME	DATE DUE